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October
1950

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Dr. Gillette Hayden
for her pioneer efforts and
selfless devotion in behalf
of periodontia and the
American Academy
of Periodontology

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THE JOURNAL OF PERIODONTOLOGY is published to serve as the professional journal of periodontia; to keep dentists in touch with research being carried on in this field and related sciences; to stimulate greater interest in the supporting tissues of the teeth and their relation to oral and general health.

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Thirty-fourth Annual Meeting

THE AMERICAN ACADEMY OF PERIODONTOLOGY

October 26, 27, and 28, 1950

Atlantic City, N. J.

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Chicago, Illinois

Chairman of the Program Committee for the thirty-fourth Annual Meeting of the American Academy of Periodontology held in the Claridge Hotel, in Atlantic City, N. J. October 26, 27, 28, 1950.

THE JOURNAL OF PERIODONTOLOGY

OCTOBER, NINETEEN HUNDRED FIFTY

A Clinical Experiment Concerned with the Problem as to Whether the Chemical Agent, Urea Hydrogen Peroxide, Influences or Hastens Tissue Repair*

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INTRODUCTION

The Rationale of Oxygen Therapy in the Treatment of Chronically Inflamed Gingival Tissue

THIS paper deals with one phase of the treatment of periodontic lesions, namely, the reduction of gingival inflammation through the local administration of oxygen. This method of treatment dates back to about 1913, when it was first introduced by Dunlop in the form of a medicated molecular oxygen.[1, 2, 3] The gas was contained in a tank under pressure and the vapor mixture was blown into the gingival tissue through an insufflation needle. Bubbles of oxygen penetrated the epithelium and connective tissue to a considerable distance from the site of application, a condition also observed by Orban in his nascent oxygen studies.[4] This method of therapy has been widely used, and has been proved of value in periodontal treatment. Artificial oxygenation was thought to produce a localized hyperemia and increased circulation, thus improving tissue oxidation.

Various oxygen-liberating substances have been used in the treatment of periodontal diseases. They may be divided into drugs which liberate oxygen on contact with catalase from the tissue fluids and tank oxygen. According to Orban,[4] the former type of compound, of which H_2O_2 is the most common, yields "atomic" or nascent oxygen which has two free electrons to the molecule available for oxidation and is in the monomeric form, O_2. Tank oxygen has its molecular structure in polymerized form $(O_2)_n$, and does not have the oxidizing ability of the former.

In 1942 Orban[5] reopened the subject by an investigation in which he used atomic oxygen in the form of 30 per cent hydrogen peroxide (Superoxol Merck). He observed that through its application, a complete change was brought about in the formerly chemically unreactive, inflamed tissue, "rendering it capable of active tissue reaction, mainly in the direction of tissue regeneration." He found that there was a reduction in the number of plasma cells;

*Presented at the American Academy of Periodontology meeting in San Francisco, October, 1949.

From the College of Dentistry, Section of Oral Pathology, University of California, San Francisco, and supported in part by the Barnes-Hind Research Fund.

new fibroblasts and capillaries developed. It was observed clinically that after treatment, "the formerly swollen, bluish-red gingival papillae and gingival margins were a bright pink in color and the swelling had subsided completely." Orban offered the explanation that the nascent oxygen accelerated local tissue oxidation because of increased circulation and the direct action of the oxygen. With a change in local oxidation, the acidity of the inflamed area was reduced, and as a result a repair of the tissue occurred.

The Superoxol method of treatment used by Orban in the oxygenation of gingival tissues was tried at the University of California College of Dentistry, and clinically his results were confirmed. However, Superoxol possesses several features which would cause it to be questioned as an acceptable therapeutic agent. It is not stable at a pH which is compatible to normal tissue, being in the range of pH 1.5 to 2.5, and requires the addition of a buffering agent before it can be used. In certain instances, it seemed to increase the inflammatory reaction but did not in any instance produce a necrosis of tissue as indicated by the manufacturer's instructions.

It seemed advisable, therefore, to find an agent with a higher pH which would be more compatible with tissue, and to incorporate other features such as a wetting agent which would promote greater absorption and, if possible, another substance which would render decomposing proteins non-toxic. These factors were obtained from a compound containing as its chief ingredients urea peroxide and sodium lauryl sulphate; and this study was carried out to determine the value of this commercial urea peroxide product when used to augment treatment of periodontal lesions by subgingival curettage.

MATERIAL AND METHOD

Material for the experiment consisted of biopsies taken from the gingival crest tissue with Orban gingivectomy knives on both control and experimental halves of the mouth at 7, 14, 21, and 28 days to check tissue changes. Both control and experimental halves received thorough subgingival curettage on the first day. Thereafter, no further treatment was given the control tissue other than home toothbrush massage. The experimental half received the chemotherapy on the first day and subsequently at each sitting immediately following biopsy. An effort was made to prevent the solution from spreading to the control tissue by using an aspirator and blocking off the opposite half of the mouth with cotton rolls.

The urea hydrogen peroxide was made up in concentrated solution using one teaspoonful of the drug (powder) to 5 cc. of water. The solution was administered with a 10 cc. Luer syringe through a curved blunt 22 ga. steel needle. It was injected into the gingival sulci and pockets by inserting the needle and using adequate pressure on the plunger. The excess solution was aspirated immediately.

Assay of Commercial Urea Hydrogen Peroxide Compound Used:*

Urea	59.0%
Hydrogen Peroxide (available)	16.91%
Sodium lauryl sulphate	2.0%
Sodium chloride	1.0%
Sodium sulphate (anhydrous) (stabilizing agent)	22.0%

This drug has a pH of about 4.5 when put into solution. The basic ingredient, urea peroxide, liberates approximately 33 per cent nascent oxygen and 65 per cent urea when dissolved in warm water, thus furnishing a high concentration of nascent oxygen without the irritating properties of other similar compounds. The high percentage of urea in the compound is an aid in the denaturation of the decomposing proteins about the pockets, in this way rendering the protein debris less toxic. The nascent oxygen serves to supply the oxygen necessary to restore the cells in the injured

*This compound should be kept under refrigeration in order to maintain its stability if storage temperatures above 70° F. are encountered.

area to normal oxidative function. The sodium lauryl sulphate is a wetting agent lowering the surface tension of the solution and thus allowing better penetration and dispersal.

The biopsies were taken from the gingival crest parallel to the long axis of the tooth. They were immediately immersed in 10 per cent neutral formol solution, fixed for 24 hours, run from 70 per cent alcohol through to absolute alcohol in eight hours, then into alcohol-ether and on into celloidin. The tissues were embedded in celloidin and then sections six microns thick were cut, stained and mounted.

Three different staining methods were used to determine tissue changes. Iron hematoxylin-aniline blue (Koneff's I.H.A.B.) was used to show mitotic figures and give contrast between intra- and extracellular elements; Masson's trichrome stain to show mitosis, connective tissue proliferation, keratinization of epithelium, and round-cell infiltration; and hematoxylin and eosin counterstain for general orientation.

DISCUSSION

A previous preliminary study was conducted in the University of California Dental Clinic by Drs. E. F. Johnson and J. Nuckolls, and served as a pilot experiment. This work was performed under clinical conditions on four humans ranging from eighteen to forty-two years of age. Nutritional and dietary surveys were made on all patients and the necessary corrections in their dietary regime were made to ensure an adequate food intake. Patients with extensive periodontal lesions involving both sides of the dental arch to about the same degree were selected in order to provide both control and experimental sides in the same mouth. The control side of the mouth received one treatment of subgingival curettage with no further therapy. The experimental side received the initial curettage, and, in addition, the urea hydrogen peroxide product which was administered subsequent to each weekly biopsy taken.

The experiment herein reported was carried on in the same manner, except that the upper arch was used as the control and the lower arch used as the experimental half of the mouth.

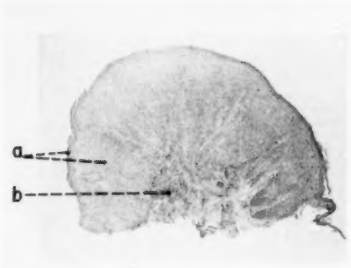
Tissues removed in the initial experiment, as noted above, were taken from the gingival crest and were sufficiently small to permit weekly biopsies to be taken from different parts of a selected area until the end of the experiment without denuding the alveolar crest. Results showed that usually the area biopsied had completely covered over with new epithelium in one week, but sometimes a small notch was present on the crest. This indicates that very rapid filling in with connective tissue elements and epithelium is possible in gingival tissue and that the biopsy effect is important,[6] i.e., sectioning of the tissue stimulates proliferation.

In order to get away from the biopsy effect, the tissue sections were taken far enough apart to eliminate this reaction, so each successive biopsy was removed one embrasure to the mesial of the one previously taken.

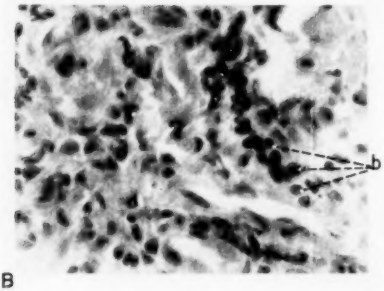
The general cleansing effect may be briefly mentioned. It is recognized by many periodontologists that cleaning up the debris and improving the hygiene in one area of the mouth seems to improve the general appearance of the tissue of the rest of the mouth. In addition to that, the product used for this experiment contains a wetting agent which enables the material to spread rapidly, thus partially overcoming all efforts to localize its action. These are shortcomings which are impossible to overcome unless a statistical project be undertaken in which several hundred mouths could be controls and several hundred others experimental.

BIOPSY FINDINGS

Case No. 1. Both control and experimental biopsies taken on the first day of the experiment show large numbers of



A

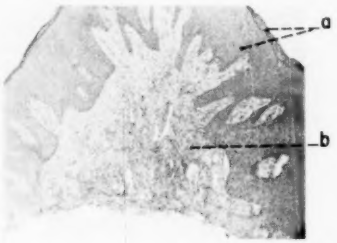


B

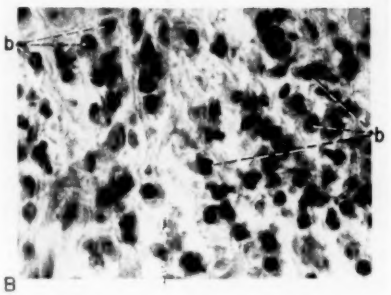
Fig. 1. A, Photomicrograph of 1st day control gingival biopsy with chronic gingivitis, Masson × 60. The epithelium (a) has a poorly developed corneous surface layer and the connective tissue (b) contains numerous round cells. B, Connective tissue of 1st day control gingival biopsy, Masson ×1000, showing plasma cells (b).

round cells in the connective tissue (Figs. 1 and 2, A and B (b)), blood vessels are numerous and enlarged, and fibroblasts are few in numbers. Connective tissue fibers are quite sparse in the experimental tissue. The epithelial pegs are short with mitotic activity in the basal layers at a minimum, and a poorly developed corneous surface layer (Figs. 1 and 2, A(a)). After a seven-day period, the numbers of round cells decrease but are still quite numerous in both control and experimental tissues, the decrease being greater in the treated tissue (Fig. 3, A and B (b)). Both control and experimental tissues are quite hyperemic. Enlarged blood vessels are shown (Fig. 3, B(c)). Connective tissue fibers are sparse and irregular in the control tissue. Round cells are still quite numerous in the 14-day control tissue (Fig. 4, A (b)), blood vessels (c) are still enlarged and fibroblastic activity is increased as shown at (d). In the experimental tissue at 14 days (Fig. 4, B), there is a further reduction in the number of round cells (b), new fibroblasts (d) appear in greater numbers than in the control tissue and the density and tone of the connective tissue fibers is increased, hyperemia (c) is still evident and mitosis is active in the basal cell epithelial layers.

At 21 days the numbers of round cells in the connective tissue of the control biopsy (Fig. 5, A (b)) have greatly decreased, but they are still numerous, mitotic activity is increased in the epithelium, blood vessels (c) are still dilated and fibroblasts (d) are



A



B

Fig. 2. A, Photomicrograph of 1st day experimental gingival biopsy with chronic gingivitis, Masson × 60. The epithelium (a) has a poorly developed corneous surface layer and the connective tissue (b) shows dense round cell infiltration. B, Connective tissue of 1st day experimental gingival biopsy, Masson × 1000, showing plasma cells (b). Connective tissue fibers are quite sparse.

more numerous with increased fiber development. The experimental tissue (Fig. 5, B) has very few round cells present, mitosis is still active with a thickening of the corneous layer of the epithelium and lengthened epithelial pegs. New blood vessels are present. Fibroblasts (d) are quite numerous and connective tissue fibers are dense and wavy, indicating a return of lost tissue tone.

The 28-day control tissue reveals a decrease in mitotic activity in the basal cell layers of the epithelium with a poorly developed surface layer (Fig. 6, A(a)). Round cells (Fig. 6, A and B(b)) in the connective tissue are fairly numerous, there is decreased hyperemia and fibroblasts (Fig. 6, B(d)) appear to be about as plentiful as in the 21-day control tissue, but with increased fiber development. Fibers are straightened, which indicates a lack of tissue tone. The experimental tissue shows further improvement. The round cells have disappeared, fibroblasts (Fig. 7, B (d)) are numerous and connective tissue fibers are wavy which indicates good tone. They are also more abundant than in the 21-day experimental tissue. The epithelium (Fig. 7, A(a)) shows long irregular pegs and a well-developed corneous layer.

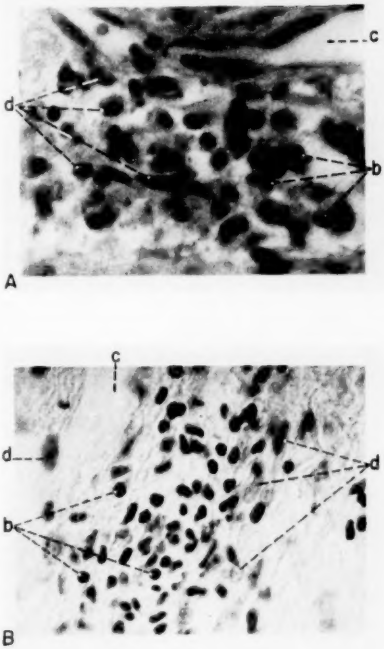


Fig. 4. A, Photomicrograph of connective tissue of 14 day control gingival biopsy. Masson × 1000. Plasma cells (b) are still abundant, blood vessels (c) are engorged and a few new fibroblasts (d) are present. Connective tissue fiber arrangement is quite irregular. B, Connective tissue of 14 day experimental gingival biopsy, Masson × 1000. Plasma cells (b) are not as numerous and new fibroblasts (d) are more plentiful than in the control biopsy with an increase in the number of fibers. Enlarged blood vessels (c) are still present.

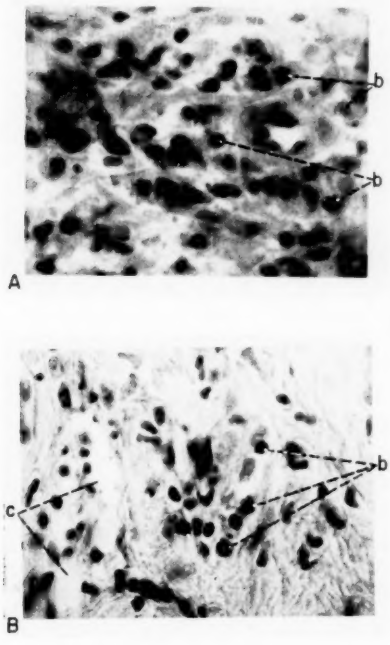


Fig. 3. A, Photomicrograph of connective tissue of 7 day control gingival biopsy. Masson × 1000. Plasma cells (b) are still numerous. Connective tissue fibers are sparse and irregular. B, Connective tissue of 7 day experimental gingival biopsy. Masson × 1000. Plasma cells (b) are not as numerous as in the control biopsy, enlarged blood vessels (c) are visible, and connective tissue fibers are more abundant.

*Case No. 2**

1st day control biopsy

The C. T. is hyperemic, round cells almost obscure the field, and there are few C. T. fibers and fibroblasts. The epithelial pegs are short, the corneous surface layer is poorly developed and the epithelium is thin.

1st day experimental biopsy

The C. T. field contains large numbers of round cells, enlarged blood vessels and few connective tissue fibers and fibroblasts. The epithelium has poorly developed pegs and cornified surface layer.

7th day control biopsy

The round cells have decreased considerably, fibers are more abundant and hyperemia is still present. The epithelial pegs are short and the surface is fairly well cornified.

7th day experimental biopsy

Round cells are more numerous than the 7th day control tissue. C. T. fibers are sparse. There are few fibroblasts and the tissue is hyperemic. The epithelial pegs are short, there is a poorly developed corneous layer and the epithelium is thin in certain areas.

14th day control biopsy

Round cells are numerous, C. T. fibers appear about the same as at 7-day control and blood vessels are dilated. The epithelial pegs are short and the corneous surface layer is very thin.

14th day experimental biopsy

There is a decrease in the number of round cells, the C. T. fibers are more dense with new fibroblastic activity. The tissue is still hyperemic. The epithelial pegs are short and the corneous surface layer is better developed.

21st day control biopsy

Round cells are still fairly numerous. New fibroblasts appear and C. T. fibers are increased in number. Some new capillaries are present. The epithelial pegs are lengthened and the corneous layer is better developed (not equal to 14-day experimental). There is increased mitotic activity in the epithelium.

21st day experimental biopsy

There are few round cells present. C. T. fibers are dense and a few new capillaries appear. The epithelial pegs are long and there is a thick corneous layer. There are numerous mitotic figures in the basal cell layers of the epithelium.

*In order to prevent the discussion of the cases from being too prolix, Case No. 1 is presented in essay form with accompanying photomicrographs at the various stages at which the biopsies were taken. The other case findings are given in outline form and the data alone are presented.

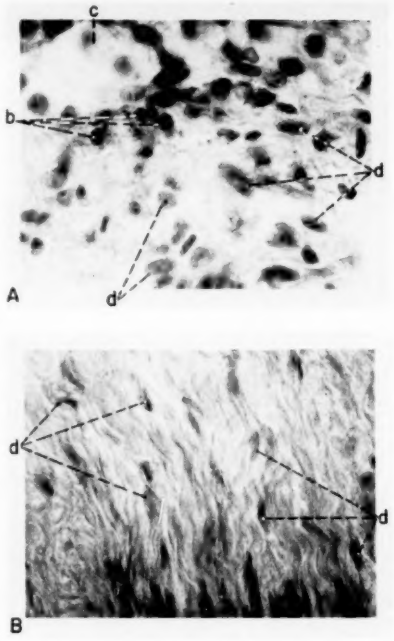


Fig. 5. A. Photomicrograph of connective tissue of 21 day control gingival biopsy, Masson × 1000. There is a great decrease in the number of round cells (b), blood vessels (c) are still enlarged, and fibroblasts (d) are more numerous with greater development of connective tissue fibers. B. Connective tissue of 21 day experimental gingival biopsy, Masson × 1000. Numerous fibroblasts (d) appear in the field with an abundance of dense wavy connective tissue fibers indicating good tissue tone.

28th day control biopsy

Round cells are still fairly numerous; C. T. fiber development is the same as in 21-day control tissue. Epithelial pegs are lengthened and the corneous layer is equal to 21-day control.

28th day experimental biopsy

There are very few round cells present, with numerous fibroblasts and dense fibers. A few new blood vessels are evident. The epithelium is well developed with long pegs and a well-cornified surface. Mitosis is active in the basal cell layers.

Case No. 3

1st day control biopsy

The connective tissue field contains a large number of round cells, is hyperemic and has few fibers present. The epithelial pegs are short and the surface is poorly cornified.

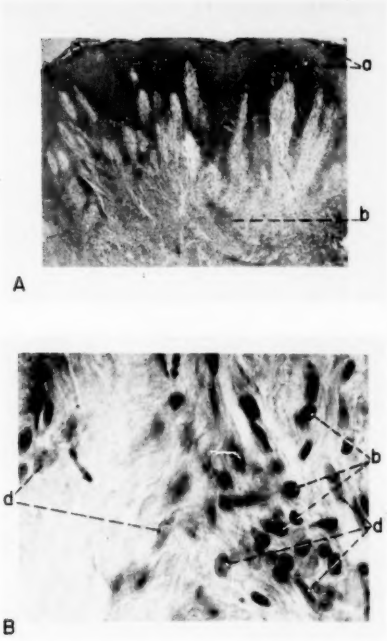


Fig. 6. A, Photomicrograph of 28 day control gingival biopsy, Masson × 60. The epithelium (a) has a poorly developed corneous surface layer. The connective tissue (b) still contains quite a few round cells. B, Connective tissue of 28 day control gingival biopsy, Masson × 1000, showing round cell (b) infiltration, fibroblasts (d) and improved fiber development.

1st day experimental biopsy

The C. T. appears about the same as the control biopsy. The epithelial pegs are short and there is no corneous surface layer.

7th day control biopsy

The C. T. is still hyperemic, there are large numbers of round cells and few fibers are present. The epithelial pegs are short and the corneous layer is poorly developed.

7th day experimental biopsy

There are large numbers of round cells in the C. T., the tissue is hyperemic and fibers are more numerous. The epithelial pegs are lengthened and a thin corneous layer is present.

14th day control biopsy

Large numbers of round cells are present, fibers are more numerous, and hyperemia is reduced in the C. T. The epithelial pegs are short and the corneous layer is slightly thicker.

14th day experimental biopsy

There is a great decrease in the number of round cells, there is new blood-vessel formation and the number of fibroblasts has increased. The epithelial pegs are lengthened with increased mitotic activity in the basal layers.

21st day control biopsy

There is a decrease in the number of round cells, fibroblasts have increased and new blood vessels are present. In the epithelium there is an increase in mitosis in the basal layers; the corneous layer is the same as the 14-day control.

21st day experimental biopsy

The number of round cells is further decreased. Fibroblastic activity is greatly increased, fibers are more dense and a few new blood vessels are present. The epithelial pegs are greatly lengthened with increased mitotic activity in the basal cell layers and a thickened corneous layer.

28th day control biopsy

C. T. fibers are more abundant, round cells are still fairly numerous and a few blood vessels are present. The pegs are lengthened, the corneous layer is thickened and there is some mitotic activity in the basal cell layers.

28th day experimental biopsy

The round cells are almost completely absent; numerous fibroblasts and dense fibers are present with a few small blood vessels. The epithelial pegs are long with increased mitotic activity. The corneous layer is much thicker than the control tissue.

Case No. 4

1st day control biopsy

The C. T. contains large numbers of round cells, is hyperemic and has few C. T. fibers and fibroblasts. The epithelium has a poorly developed corneous layer and short pegs with little mitotic activity.

1st day experimental biopsy

The appearance of this tissue bears the same description as the first day control.

7th day control biopsy

A large number of round cells, few fibroblasts and tissue hyperemia can be seen. Epithelial pegs and corneous layer are poorly developed.

7th day experimental biopsy

There is a slight decrease in the number of round cells; fibroblasts and C. T. fibers are more numerous. Epithelial pegs are slightly lengthened and the corneous layer is still poorly developed.

14th day control biopsy

C. T. examination reveals a considerable decrease in the number of round cells and decreased hyperemia. Fibroblasts and fibers are increased in number.

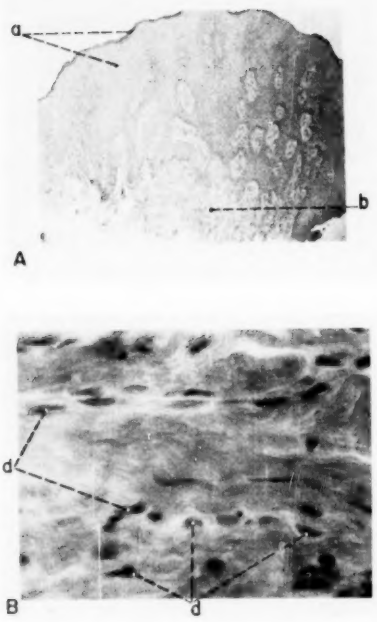


Fig. 7. A. Photomicrograph of 28 day experimental gingival biopsy. Masson × 60. The epithelium (a) has a well developed stratum corneum and long pegs are visible. The connective tissue (b) is devoid of round cells. B. Connective tissue of 28 day experimental gingival biopsy. Masson × 1000, showing fibroblasts (d) and dense wavy fibers.

The epithelial pegs are lengthened and the corneous layer is thickened.

14th day experimental biopsy

There is a great decrease in the number of round cells; new blood vessels, fibroblasts and fibers are observed. Epithelial pegs are lengthened and the corneous layer is as thick as in the 14-day control biopsy.

21st day control biopsy

Round cells are further decreased, new blood vessels are present and there is an increase in fibroblastic activity. The epithelial pegs are lengthened and the corneous layer is slightly thickened.

21st day experimental biopsy

Fibroblasts are increased in number and there are a few enlarged blood vessels. Round cells have further decreased. Epithelial pegs are long and the corneous layer appears the same as the control.

28th day control biopsy

Round cells are slightly numerous; there is an increased number of fibroblasts and a few new vessels in the C. T. The epithelium has lengthened pegs with an increased thickening of the corneous layer. Mitotic activity is reduced.

28th day experimental biopsy

Fibrosis in the C. T. is increased and the fibers are dense; a few small blood vessels are present and round cells have almost completely disappeared. The epithelium appears normal with long pegs and a well-developed corneous layer. Mitosis is still active in the basal cell layers.

SUMMARY AND CONCLUSIONS:

The gross clinical changes in the appearance of the gingival tissue were marked on the experimental half of the mouth. After the injection into the pockets with urea hydrogen peroxide, the cyanotic appearance of the tissues disappeared within a few minutes and the tissue appeared pink in color and remained so throughout the period of observation.

Histological examination revealed a definite progressive change in both the control and experimental tissues. Before treatment, the biopsies showed the connective tissue underlying the epithelium to be almost obscured by the mass of round cells. The capillaries were numerous and dilated. There were few fibroblasts in evidence. One week after treatment, both the control and experimental biopsies exhibited a definite decrease in round cells, the treated tissue showing the greater change. Both control and experimental tissues at 28 days showed an increase in mitotic activity in the basal cell layers of the epithelium, the increase being greater in the experimental tissue with a greater thickening of the keratinized surface layer and a lengthening of the epithelial pegs. Biopsies taken from the experimental half of the mouth up to 28 days demonstrated the disappearance of round cell infiltration and the proliferation of connective tissue elements resulting in the appearance of new capillaries and greater fibroblastic activity producing an increase in tissue fibrosis. There was also an improvement in tissue tone as evidenced by a waviness of the C. T. fibers.

From this study one can conclude that when nascent oxygen is applied locally to inflamed gingival tissue, the inflammatory process is more rapidly reduced. This may be observed clinically as well as histologically. The disappearance of the plasma cells and the increased mitotic activity in both epithelium and connective tissue with resultant prolifieration of cellular elements are probably the result of increased tissue oxidation brought about by the local oxygen therapy.

The author would like to acknowledge the contribution of Doctor E. F. Johnson in running the pilot experiment, and to express his appreciation and indebtedness to Doctor James Nuckolls for his counsel and criticisms in conducting this study, and to Doctor H. E. Frisbie and Mrs. B. Stutzman for assistance in photomicrography, and to Mrs. H. Whitaker for secretarial aid.

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Calculus Formation*

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THE available evidence indicates that the inorganic constituents of oral calculus are derived, certainly in the main and probably almost entirely, from saliva. Calculus-like deposits have been successfully produced *in vitro* from saliva[1, 2, 3, 4] and from solutions made up to simulate saliva.[1] Ordinarily, the saliva contains the principal constituents (calcium and phosphate) in relatively high concentrations. Upon exposure of the saliva to oral conditions, certain forces come into play which lead to the precipitation of the calculus mineral. The mechanisms, i.e., exactly what these forces are and how they act, have not been fully elucidated, although many theories have been proposed to explain these phenomena. None of the theories, however, has been successful in accounting for the observed individual differences in the degree of calculus formation or for the variations in the accumulation on different tooth surfaces in the same mouth.

Aside from the problem of precipitation, there is the associated problem of how the precipitates are held together and how they are attached to the tooth surface to present what is clinically dental calculus. One might thus visualize the process of calculus deposition as consisting of at least two closely associated phases, precipitation and attachment.

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There is general agreement that bacteria play an important role in the attachment of calculus.[2, 4, 5] Histologic examination of sections of calculus shows networks of filamentous microorganisms that seem to enmesh the inorganic precipitates.[6, 7] The exact identity of these organisms is still a matter of uncertainty but their importance in holding the precipitate together and fixing it to the tooth surface is generally accepted.

There is, however, no such agreement with regard to the factors thought to be responsible for producing the precipitation of the mineral from saliva. The major theories to explain this phenomenon may, for convenience, be divided into three main groups, namely, the bacteriological, enzymatic, and physico-chemical theories. These designations are self-explanatory, and are based upon which one of the three factors is emphasized in explaining the processes leading to the precipitation of the inorganic substances from saliva.

Proponents of the bacteriological theory hold that the microorganisms in the mouth, through their growth and metabolism, produce conditions which lead to the precipitation of those elements which enter into the structure of calculus. As early as 1886, Gallippe[8] stated that the microorganisms found in salivary tartar were not there by accident but, instead, were the agents which produced the concretions. This point of view has been elaborated upon by many subsequent workers, including Goodrich and Moseley,[9] Bulleid,[7] Naeslund,[2] and Kofoid and Hinshaw.[3] Naeslund[2] identified the responsible organisms as belonging to the genera *Leptotrichia* and *Actinomyces*. It was postulated that these bacteria produced a local alkalinity and a decomposition of the salivary proteins in their immediate vicinity. The changes in the local environment, in turn, caused the precipitation of the calcium salts from the saliva. The ability of these organisms to produce such changes has not, however, been clearly demonstrated.

In 1929, Adamson[10] showed the presence of the enzyme phosphatase in human gingival tissue. This enzyme was found to be capable of acting upon certain 'phosphate-containing complexes' in saliva leading to the 'liberation and deposition of free inorganic phosphate.' When silk threads, previously soaked in gingival extract (dog tissue), were suspended in saliva containing the gingival extract, concretions were deposited on the threads in seven days. When no extract was present, or when the extract had been boiled, no deposits were seen. Adamson[10] therefore postulated that an enzyme, liberated from the gingival tissue by trauma, acts upon the saliva to form tartar. Smith,[11] in the following year, reported that the enzyme had an optimum pH of 5.5 and was present in the desquamated epithelial cells of the saliva. He supported Adamson's[10] contention that the action of this enzyme on the salivary organic phosphates can lead to the production of calculus in the mouth.

Up to the present time, however, there has been no demonstration that such a mechanism does operate within the mouth, or indeed, that the intervention of an enzyme such as phosphatase is necessary for the production of calculus, either *in vitro* or *in vivo*. Neither has it been shown that the calcific deposits produced by Adamson[10] on the silk threads are, in any way, similar to oral calculus. Zander[12] was unable to confirm Smith's[11] findings that phosphatase was present in the gingival epithelium; and Saltzmann[13] found that the degree of phosphatase activity of mixed saliva was not related to the presence and quantity of calculus in the mouth. The phosphatase theory probably should be discarded.

The physico-chemical group of theories considers such diverse factors as CO_2 loss, NH_3 production, denaturation or decomposition of proteins, precipitation of proteins, changes in surface tension and changes in colloidal properties. All these factors have one point in common: they are all believed to lead to a decreased solubility in saliva of

the calcium salts that enter into the construction of calculus. Kirk[14] was of the opinion that any agency which would 'eliminate the acid element' or 'increase the basic content' of the saliva would lead to a precipitation of those salts. Especially important, he thought, was the loss of CO_2 from the saliva following its secretion into the mouth. A similar point of view was advanced by Moore,[15] Pickerill,[16] and others. Developing this idea of CO_2 loss, Rapp[17, 18] adapted Fosdick and Starke's[19] 'critical pH' to the interpretation of calculus formation. It was postulated a) that for each ratio of calcium and phosphorus in the saliva there is a critical pH above which precipitation will occur, and b) that the elevation of the pH of the secreted saliva above the critical level is brought about through the loss of CO_2.

Another possible way of increasing the alkalinity of the secreted saliva is by the production of ammonia. This aspect of the problem has not received as much attention, and the extent to which ammonia may be produced in the mouth and whether it may lead to the precipitation of the calcium salts is unknown, although Frank[20] has reported that the ammonia content of the saliva was directly related to the individual's tendency to form calculus. Such a relationship can hardly be considered as demonstrating cause and effect.

A different approach was made by Prinz.[1] He postulated that as the saliva stagnates, the colloidal proteins become concentrated at the surface and the protective colloidal action of the proteins is lost. The calcium salts then precipitate and are carried down with the 'colloidal stroma' until they come to rest, attached to a tooth surface.

Karlström and Miller[21] have shown that the proteins do, indeed, decrease the amount and rate of calcium precipitation following CO_2 loss from mixtures made in various proportions of $Ca(OH)_2$, Na_2HPO_4, NH_4OH and $NaHCO_3$. They were not able to show, however, that allowing the mixture to stand for 24 hours, during which 'stagnation' could presumably occur, would lead to the sedimentation of a precipitate. In addition, it is difficult to imagine how appreciable 'stagnation' of saliva could exist under the usual oral conditions. Certainly, the frequent finding of heavy deposits on those tooth surfaces contiguous to the orifices of the salivary ducts, where one may expect relatively rapid salivary flow and the least opportunity for stagnation, would seem to argue against the concept of stagnation as being an important factor in calculus formation.

From this brief review of the current theories of calculus formation, it is evident that there is no general agreement regarding the mechanism. Beyond the opinion that the phosphatase theory as stated is untenable, there is as yet insufficient evidence to support any one theory to the exclusion of the others. Insofar as the bacteriological and physico-chemical theories are concerned, the same mechanism of precipitation of the calcium salts is postulated for both. An important difference lies in the specific factors held responsible for the decreased solubility of the calcium salts. The bacteriological theory, as propounded by Naeslund,[2] requires the metabolic activity of bacteria to produce a local alkalinity and consequent decreased solubility. The physico-chemical theories, on the other hand, do not require the intervention of bacteria, but hold that the 'normal' alterations in the physical and chemical properties of saliva, such as the loss of the colloidal protective action of the proteins, or the escape of CO_2, are in themselves sufficient to lead to the precipitation of the calcium salts. The evidence presented below supports the view that the precipitation of the calculus mineral may follow the loss of CO_2. It does not, however, exclude the participation of other factors in calculus formation.

Chemical Composition of Calculus. Considering the problem of calculus formation from a chemical angle, there are two basic problems: 1) what is calculus, chemically; and 2) what are the laws governing its formation? It seems logical to review the evi-

TABLE 1

*Analyses of Calculus**

H_2O Organic Matter (%)	Inorganic Matter (% inorganic)					Source
	Ca	P	Ca/P	CO_2	Mg	
17	30.7	16.8	1.83	1.39	1.0	Subgingival (22)
25	36.8	20.5	1.79	0.28	Tr.	Salivary (23)
21	35.9	16.8	2.14	5.3	—	Wharton's Duct (24)
26	38.1	17.6	2.16	7.4	0.5	Human (24)
13-23	37.7-39.0	18.7-19.1	2.01	1.38-2.39	0.4-0.8	Submaxillary (1)
			Average Analyses of Tooth Tissues			
6	37.6	18.4	2.04	3.05	0.7	Enamel
43	35.5	16.7	2.12	2.94	1.4	Dentine
75	36.7	16.0	2.29	5.86	0.5	Bone (26)
			Analyses of Horse Calculi			
13	39.9	0.6		57.9		(24)
8	39.5	0.7		57.3		(25)

*Other elements in trace amounts in calculus: K, (P)
Na, (P)
SO_3 (P) (Pr = 0.17 as H_2S)
Cl (P)
SiO_2 (P) (Gh) (Gh—$CaSiO_3$ = 0.08)
Fe, (0.02—G&M) (0.01—Gh)

dence on the chemical nature of calculus before proceeding with a discussion of its solubility.

In the accompanying table (Table 1) several analyses of calculus made by different investigators have been compiled. The calculi came either from the surface of teeth or were duct or gland stones. Most samples came from human patients; for comparison, the data of two investigators are included who reported analyses of horse calculi. In the lower part of the table, selected average analyses have been put down for tooth, enamel and dentine separately, and for bone.

The percentages of water (moist weight basis) are low, viz., around 1% for the horse calculi, but range from 6 to 20% for the other samples. Enamel also has a low percentage of water (2.3%), dentine has a water content in the range of values recorded for calculi (13%) while bone contains considerably more water, in some samples as much as 50%. The organic matter of calculi ranges from 6 to 15% (moist weight basis). These values are much larger than the organic content of enamel (3.9%) but lower than that of dentine or bone (around 30%). It is surprising to find more water and organic matter in dentine than in calculus. The common picture of calculus includes masses of bacteria and mucin so that the high mineral content is unexpected.

When the percentages of the *inorganic* constituents are compared, two major conclusions may be drawn: first, calculi from the horse are different than calculi from man, specifically the percentage of phosphorus varies from about 17 to 20 in the human calculi, whereas two investigators report 0.64 to 0.74 per cent phosphorus in horse calculi. The principal anion of

horse calculi is CO_2; values of 57.3 and 57.9% are reported as contrasted with 0.3 to 7.4% CO_2 in human calculi. Horse calculi are crystalline calcium carbonate; X-ray studies such as those reported by Brandenberger[27] have revealed the crystals to be calcite.

The second major conclusion relates to the remarkable similarity of the inorganic composition of human calculus, enamel, dentine, and bone. For all these tissues, calcium values range from 30 to 40% (mostly from 36 to 38%). Phosphorus values range from 16 to 20% (mostly from 16 to 18%). Magnesium values range from 0.4 to 1.4% and CO_2 values range from 1.4 to 7.4% (mostly from 2 and 5%). The calcium to phosphorus ratios of the various calculi range from 2.01 to 2.16 with the exception of one sample reported by Philipp[23] to have a value of 1.8. These analyses are in complete agreement with the general principle that all calcium phosphates in the body have a 'remarkable constancy' of composition. Viewed in this light, calculus might be considered to be an ectopic calcification comparable to calcified nodules in the lung, calcific material in blood vessel walls, calcification in muscle or in other sites described in the literature.

Crystal Lattice of Calculus. Recognizing the similarity of calculus to other calcium phosphates in the body, such as tooth and bone substance, is important. It is likewise important to know that the same crystal lattice, namely, hydroxylapatite, has been found in all these calcium phosphates by the use of X-ray diffraction methods. It is thereby established that the spatial arrangement of the calcium ions relative to the phosphate ions is identical in all these tissues.

A word might be in order regarding the concept of the crystal lattice, especially since theories of solubility have been based in some cases on the geometry of solid crystals in contact with a solution. Some substances, for example, solid sodium chloride, are made up of a simple space arrangement which is represented by a cube with alternating sodium and chloride ions at the corners. In this space arrangement, each sodium ion is in 'contact with' six chloride ions and each chloride ion has the same relation to six sodium ions. From the arrangement of atoms, the whole crystal might be described as composed of doublets, each doublet consisting of a sodium and a chloride ion. The crystal therefore is a macromolecule of NaCl. When such a crystal goes into solution, sodium and chloride ions must leave alternately, and the solubility is a simple function defined by the product of the concentration of sodium ion and the concentration of chloride ion.

In the case of hydroxylapatite, the calcium phosphate of the body, a great deal of confusion has arisen because chemists have tried to give a simple formula by which the mineral phase could be described. It would indeed be useful if some simple formula did apply. Unfortunately, the crystal lattice of the calcium phosphate found in calculus, in tooth and in bone substance is not a simple one. This lattice has been given the name 'hydroxylapatite.' The lattice is highly complicated; hydroxyl, phosphorus, calcium and oxygen atoms are set in an intricate pattern that has been carefully worked out by X-ray crystallographers. The time honored formula—tricalcium phosphate, $Ca_3(PO_4)_2$—is not applicable. Even a searching study of the space arrangement of these atoms does not reveal a grouping of three calciums and two phosphates anywhere. There is not the slightest resemblance to a macromolecule formed by a repetition of groupings of $Ca_3(PO_4)_2$; in fact this simple formula has nothing whatsoever to do with the crystal lattice. Nor has any other simple formula. This calcium phosphate is properly described as *the hydroxylapatite lattice* which refers to a particular and exact and defined arrangement of calcium, phosphorus, oxygen and hydrogen atoms. It is not possible to describe this lattice by a simple chemical formula.

Certain properties of the apatite lattice

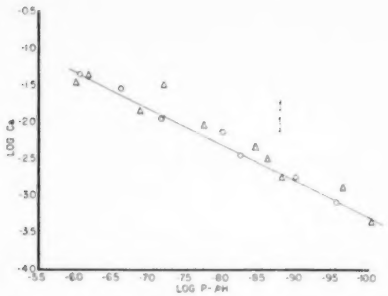

Fig. 1. △-enamel in saliva. ○-hydroxylapatite in saliva. Data of Ericsson.

also have been reported for calculus. Trömel and Möller[28] in 1932 showed the hydroxylapatite precipitates (prepared *in vitro*) having a low calcium to phosphorus ratio would undergo a transformation of the lattice to that of beta tricalciumphosphate after ignition at 900° C. Brandenberger[27] found that certain samples of calculus would also exhibit this same transformation depending on the calcium to phosphorus ratio. It will be shown later that the solubility characteristics of hydroxylapatite also probably hold for enamel and calculus.

Solubility Studies. It seems almost self evident that saliva ought to be saturated with respect to tooth enamel. The mineral phase of enamel is hydroxylapatite, therefore saliva ought to be approximately saturated with respect to hydroxylapatite. It is, therefore, of considerable interest to find from data published recently by Ericsson[29] that the solubility of powdered tooth enamel in human saliva and the solubility of a synthetic hydroxylapatite in human saliva may be described by the same straight line (Figure 1).

In Figure 2, the calcium content, as determined by Schmidt-Nielsen,[30] of saliva samples after equilibration for various periods, usually at least several days, are plotted on a logarithm scale as ordinates against the logarithm of the phosphate concentrations of the same samples minus the final pH of the saliva. The regularity with which the experimental points fall on the line taken from Figure 1 is notable.

Significance of Log Ca vs. Log P-pH. The selection of coordinates for Figures 1 and 2 is a little unusual and a word of explanation is in order. It was recently noted (Hodge[31]) that aqueous solutions in contact with precipitates of calcium phosphate have calcium and phosphate concentrations such that when the logarithm of the calcium concentration is plotted against logarithm of the phosphate concentration, a straight line relation is found. Stated mathematically, $\frac{Ca}{P} = K_1$, or, the ratio of calcium to phosphate concentrations is a constant. Please note that this is not a solubility product, it is a ratio.

It was also found (Shear and Kramer[32]) that the pH of the solutions in contact with precipitates of calcium phosphate varies with the calcium concentration. Stated mathematically, $\frac{Ca}{H} = K_2$, or, the ratio of calcium to hydrogen concentrations is a constant.

If these two equations are multiplied, the following equation is obtained: $\frac{Ca^2}{H \times P} = K_3$, or, the ratio of the calcium concentration squared to the product of the hydrogen and phosphate concentrations is a constant. If the logarithm of this equation is taken, the following equation may be written: $2 \log Ca = \log P - pH + K_4$. This equation states that the logarithm of the calcium concentrations should vary linearly

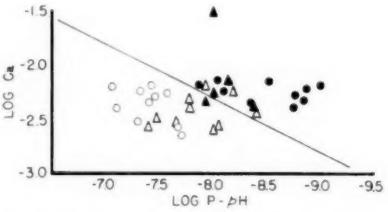

Fig. 2. Data on parotid saliva samples from students (○) and from pregnant women (△); on mandibular saliva samples from students (●) and from pregnant women (▲). Data of Schmidt-Nielsen.

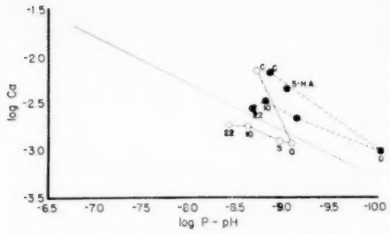

Fig. 3. Relationship of calcium and phosphorus concentrations and pH in saliva equilibrated with solid synthetic hyrdoxylapatite at various CO_2 tensions. (Sample No. 1—( ), sample No. 2 ( ). Trend line taken from Figure 1.

with the logarithm of the phosphate concentration minus the pH. Consequently the straight line in Figure 1 was expected when the log Ca is plotted against log P–pH. From the equation it is predicted that the slope of the line should be 2; it is interesting to find from comparing two points (e.g., ordinate – 1.5, abscissa – 6.3; ordinate – 3.0, abscissa – 9.4) that the slope closely approximates 2.0.

The triangles in Figure 1 represent the concentrations of calcium, phosphorus and hydrogen in saliva samples shaken with powdered enamel. If enamel is principally hydroxylapatite then these data should represent the solubility of hydroxylapatite in saliva. Ericsson[29] has also given data indicated by the circles (Figure 1) on the calcium, phosphorus and hydrogen concentrations in saliva samples shaken with a synthetic hydroxylapatite. As was expected, the solubility of hydroxylapatite describes with exactness the solubility of enamel in saliva.

Unfortunately, there are no data available on the solubility of calculus in saliva. Since calculus also contains hydroxylapatite as its principal mineral, it is predicted that the solubility of calculus in saliva will also be described by the straight line in Figure 1.

Relative Saturation of Saliva. Schmidt-Nielsen[30] recently published a study of the chemical composition of saliva in which she included data on the calcium, phosphorus and hydrogen concentrations. In Figure 2, some of her data have been recalculated and plotted on the same coordinates that were used in Figure 1. She gave data on samples from students (circles) and from pregnant women (triangles). Parotid saliva (open circles and triangles) and mandibular saliva (solid circles and triangles) samples were analyzed separately. The straight line in the figure indicates the trend line of Figure 1 for the solubility of hydroxylapatite and for enamel in saliva. It is of considerable interest to find that the saliva samples all give points clustered closely around the hydroxylapatite solubility curve. In other words, all the saliva samples had calcium, phosphorus and hydrogen concentrations not far removed from saturation conditions for enamel and presumably for calculus.

A close inspection of the figure reveals an interesting selective distribution of the points depending on the origin of the saliva. All but three of the parotid saliva samples give points lying below the trend line and all but one of the mandibular saliva samples give points lying above the line. Since in general, points above the line represent supersaturation with respect to calcium and phosphorus, the mandibular salivas are almost without exception supersaturated with respect to hydroxylapatite. Conversely, since points below the line represent salivas unsaturated with respect to calcium and

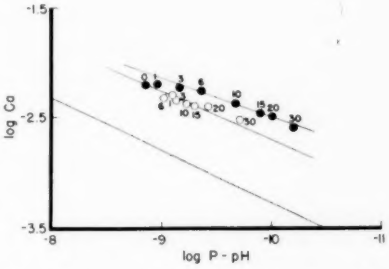

Fig. 4. Relation of calcium and phosporus concentrations and pH to loss of CO_2 into room air in periods up to 30 minutes. (Sample No. 1 ( ); sample No. 2—( ). Trend line is taken from Figure 1.

TABLE 2
Effect of Incubating Saliva with Synthetic Hydroxylapatite and CO_2

Condition	CO_2	*Calcium*			*Phosphorus*			*pH*	*log P-pH*
	mM/l	*mM/l*	*gm/100 ml*	*log*	*mM/l*	*gm/100 ml*	*log*		
Sample No. 1									
Control	12.65	1.74	0.00696	−2.16	5.00	0.0155	−1.81	6.9	−8.71
0.0% CO_2	0.72	0.29	0.00116	−2.94	3.49	0.0108	−1.97	7.1	−9.07
5.0% CO_2	8.73	0.31	0.00124	−2.91	3.58	0.0111	−1.95	7.0	−8.95
10.3% CO_2	9.59	0.44	0.00176	−2.75	3.99	0.0124	−1.91	6.7	−8.61
22.1% CO_2	14.79	0.46	0.00184	−2.74	3.93	0.0122	−1.91	6.5	−8.41
Sample No. 2									
Control	15.54	1.62	0.00648	−2.19	4.54	0.0141	−1.85	7.0	−8.85
0.0% CO_2	6.65	0.24	0.00096	−3.02	3.87	0.0120	−1.92	8.1	−10.02
5.0% CO_2	10.87	0.53	0.00212	−2.67	3.82	0.0118	−1.93	7.2	−9.13
10.3% CO_2	10.65	0.81	0.00324	−2.49	3.95	0.0122	−1.91	6.9	−8.81
22.1% CO_2	16.26	0.68	0.00272	−2.57	4.33	0.0134	−1.87	6.8	−8.67
5.0% s hydroxylapatite	12.51	1.12	0.00448	−2.35	4.73	0.0147	−1.83	7.2	−9.03

phosphorus, the parotid salivas are mostly unsaturated with respect to hydroxylapatite. These data indicate that mixed saliva samples generally are more or less saturated with respect to hydroxylapatite (enamel or calculus); that under some circumstances saliva might be unsaturated, that is, would dissolve enamel or calculus; and that under some circumstances saliva is supersaturated, that is, a calcium phosphate (hydroxylapatite) might precipitate.

Recently one of us (Leung) studied the effect of incubating saliva with CO_2 and hydroxylapatite. Two such experiments are illustrated in Figure 3 and Table 2. The control mixed stimulated saliva samples give points considerably above the solubility curve; that is, they were supersaturated with respect to hydroxylapatite. When powdered synthetic hydroxylapatite was added to the saliva and the mixture was incubated with nitrogen containing 0% CO_2, nearly all of the CO_2 was removed. At the same time a large part of the calcium and quite a little of the phosphorus was thrown out of solution. The pH became elevated. From the figure it is evident that the loss of calcium, the loss of phosphorus, and the increase in pH have been just enough to change the saliva from a condition of supersaturation to a condition approximating saturation.

In addition to incubating with 0% CO_2, aliquots of the saliva-hydroxylapatite mixtures were also equilibrated with nitrogen containing 5, 10 and 22% CO_2, respectively (Table 2). With each increase in the CO_2 concentration there was a decrease in the pH and an increase in the amounts of calcium and phosphorus remaining in solution in the saliva. As is evident from Figure 3, these changes are of such magnitude as to approach the solubility conditions for hydroxylapatite.

It is interesting to note that when no hydroxylapatite was added to the saliva the changes in calcium and phosphorus concentrations, which accompany changes in the CO_2 content, are of such magnitude as to leave the saliva still considerably supersaturated with respect to hydroxylapatite. This is illustrated in Figure 3. In this figure the specimens indicated by the solid circles marked 5 and 5-H.A. were both incubated with 5% CO_2. But only to the former specimen was synthetic hydroxylapatite added. From Table 2 it may be seen that the pH of the two specimens is identical. However, the specimen without hy-

TABLE 3
Effect of Exposing Saliva to Room Air on the Salivary CO_2, Calcium, Phosphorus and pH

Duration of Exposure (min.)	*CO_2*	*Calcium*			*Phosphorus*			*pH*	*log P-pH*
	mM	*mM*	*gm/100*	*log*	*mM*	*gm/100*	*log*		
0	14.75	1.53	0.00512	−2.21	4.52	0.0140	−1.85	7.0	−8.85
1	14.40	1.56	0.00525	−2.20	4.44	0.0137	−1.86	7.1	−8.96
3	13.26	1.45	0.00581	−2.24	4.43	0.0137	−1.86	7.3	−9.16
6	11.67	1.37	0.00548	−2.26	4.45	0.0138	−1.86	7.6	−9.46
10	10.32	1.06	0.00425	−2.37	4.21	0.0131	−1.88	7.8	−9.68
15	9.41	0.83	0.00332	−2.48	4.19	0.0130	−1.89	8.0	−9.89
20	9.11	0.79	0.00316	−2.50	4.12	0.0127	−1.90	8.1	−10.00
30	8.27	0.63	0.00252	−2.60	4.11	0.0127	−1.90	8.3	−10.20
				Sample No. 2					
0	19.60	1.18	0.00472	−2.33	4.84	0.0150	−1.82	7.2	−9.02
1	19.03	1.17	0.00468	−2.33	4.94	0.0153	−1.82	7.3	−9.12
3	18.80	1.25	0.00500	−2.30	4.90	0.0152	−1.82	7.3	−9.12
6	18.83	1.24	0.00496	−2.31	4.96	0.0154	−1.81	7.3	−9.11
10	18.48	1.06	0.00424	−2.37	4.94	0.0153	−1.82	7.4	−9.22
15	17.59	0.99	0.00396	−2.40	5.14	0.0159	−1.80	7.5	−9.30
20	16.81	0.99	0.00396	−2.40	4.94	0.0153	−1.82	7.6	−9.42
30	14.69	0.75	0.00300	−2.52	4.98	0.0154	−1.81	7.9	−9.71

droxylapatite contained considerably more calcium and somewhat more phosphorus in solution (or in suspension) than did the one with hydroxylapatite. Adding hydroxylapatite not only increased the amount of calcium and phosphorus precipitated from the saliva, but caused them to precipitate in such a way as to make the residual salivary calcium and phosphorus satisfy the solubility conditions for hydroxylapatite.

The results of recent studies into the relationship between CO_2 loss and calcium and phosphorus precipitation from saliva also illustrate this point. Stimulated mixed saliva was taken and exposed to air in an incubator for various intervals of time. The loss in CO_2, and the accompanying changes in calcium and phosphorus concentrations, and in the pH of the saliva, were measured in relation to the duration of exposure. Figure 4 shows the results of two such studies. No solid hydroxylapatite was added and no attempt was made by aeration to control the percentage of CO_2 in the atmosphere above the saliva.

It will be noticed that, in contrast to Figure 3, all the points fall considerably above the solubility curve. As the CO_2 is lost from the saliva, some calcium is precipitated in such a manner as to make the points move in a direction more or less parallel to the solubility curve. They do not move closer to the curve. In other words the changes in pH, calcium and phosphorus, which accompany the CO_2 loss, are such as to maintain the saliva in a supersaturated condition with respect to hydroxylapatite. From Table 3 it will be noticed that, actually there is very little change in the phosphorus concentration following exposure of the saliva. It is possible that the calcium is precipitated in some other form than as hydroxylapatite, e.g., as a carbonate or proteinate. It is also possible that a colloidal precipitate of hydroxylapatite is present; the amount of the colloid increases with CO_2 loss (i.e., with time) but the relation of log Ca to log P–pH (the slope) is maintained as is characteristic of hydroxylapatite. Compared to Figure 3, these curves are in the supersatu-

rated area; the solid hydroxylapatite may have presented an adequate surface on which lattice growth could keep pace with the decreasing solubility. The latter possibility is the more attractive explanation.

The data make possible a simple and reasonable description of the relations between enamel, saliva and calculus.

1. Saliva normally is approximately saturated with respect to enamel (hydroxylapatite). Enamel does not dissolve.

2. Normal saliva may be supersaturated. A considerable supersaturation is necessary before a precipitate will form, therefore, normally, calculus does not form.

3. Loss of CO_2 from saliva may increase the supersaturation of saliva to a point that precipitation occurs. If this precipitate is calculus (hydroxylapatite), the solubility relationship of the enamel-saliva system should exactly be satisfied (i.e., the saliva becomes just saturated with respect to enamel). Under such conditions enamel would *NOT* tend to dissolve, i.e., enamel decalcification (caries?) would be inhibited.

4. In unsaturated salivas, calculus would have less chance of forming even with CO_2 loss. However, such salivas would tend to dissolve enamel so that these conditions would simultaneously favor enamel decalcification (caries?) and prevent calculus formation.

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In Our Opinion

Raising the Line of Epithelial Attachment and Increasing Depth of Clinical Root

Initial Statement

By HAROLD J. LEONARD, D.D.S., B.A., *New York, N. Y.*

IT IS the property of epithelial tissue to cover and protect underlying tissues and to grow downward to envelop irritants which have penetrated beneath its surface. The periodontal pocket is formed primarily by a downward growth of the band of epithelial attachment on the root surface at the expense of the principal fibers of the periodontal membrane which have presumably become degenerated and can no longer resist replacement by epithelium. As the epithelial attachment band grows downward toward the root apex, the border toward the crown degenerates and becomes detached from the root surface, thus deepening the sulcus into a pocket. The how and why of such degeneration will not be considered at this time.

When a pocket is treated and relieved of all irritants and inflammation without appreciably disturbing the band of epithelial attachment, one of three results may occur: (1) The pocket remains as a deepened sulcus, the free gingival wall being held tightly against the tooth by healthy gingival connective tissue. (2) The gingival wall gradually shrinks down until the gingival sulcus is of normal depth. This appears to occur in persons who are extremely vigorous with the tooth brush and who probably give some gingival abrasion in the course of gingival massage. (3) The lining epithelium of the pocket attaches itself to the root surface, thus reducing the pocket to a normal or nearly normal sulcus depth without recession of the gingival margin. B. Orban contends that such reattachment of the epithelial lining of the pocket is impossible, that reattachment

of epithelium can only occur by a gradual creeping growth of the basal epithelial cells along the root surface. However, periodontists from the time of Younger around 1890 have observed such reattachment clinically and a few photo-micrographs of Harold Box published in 1924, and of William Reppeto, not published but copies of which are in the possession of the author, show it histologically.

Any of the three results enumerated above are capable as a rule of maintaining teeth safely in comfort and function for as long as the efficient home care and twice yearly treatments to eliminate debris inaccessible to the patient are kept up. Root surface curettage has been the standard procedure by most periodontists for the last fifty years.

Starting around 1920 the surgical resection of the gingival wall has been gaining popularity in the United States as a means quickly and surely of eliminating diseased tissue and reducing the gingival sulcus to a normal depth or even to zero. The operation at its best is a combination of contoured resection of gingiva, curettage of all epithelial attachment and bits of soft granulating tissue, scaling of all debris from the exposed root surfaces and packing with an antiseptic cement. The cement pack prevents the formation of excess granulation tissue masses until the cut surface is covered over with epithelium which attaches to the root surface to form a new band of epithelial attachment. It would be expected that this new band would form in a line nearer the root end than the old band which was cut away but I. Glickman (lecture at Tufts College Dental School Berkshire Conference, June 14-17, 1950) claims on the basis of experimental and histological evidence that it is crownward. A crownward line of epithelial attachment means a greater reattachment of principal fibers with formation of new cementum and a higher level of supporting bone. If such a result can be attained by surgical resection when it can not by simple root surface curettage then there is a strong argument for resection. Some of the clinical results six months after resection suggest such a crownward growth of epithelial attachment as well as marginal gingiva. I wonder if any of the discussors have any data bearing on this point.

Another method of getting a new line of epithelial attachment is that of curetting the old epithelial attachment at the time of root surface curettage and preventing by one means or another its reformation until connective tissue has built in to form a new connective tissue attachment. F. E. Beube has been working along this line. The idea seems to work in periodontal abscesses where the marginal gingiva is fairly healthy and hugs the tooth tightly. In such cases the diseased area can be opened as for an apicoectomy, curetted thoroughly, left filled with blood clot with or without foreign bone powder and then stitched tight. In such a case the epithelium does not grow down into the wound but grows over the blood clot and forming connective tissue, to the root surface where it attaches at a level far crownward from the former pocket depth. I doubt whether such a result can be made to occur when the operation is performed through the open pocket although Harold Box and others are attempting it. The difficulty is that a blood clot can not be maintained in an open pocket sufficiently intact while it is being replaced by connective tissue, to prevent epithelium from growing down to its former depth.

An interesting observation has been made by an orthodontist, Saul M. Bien of New York, some of whose cases I have seen. In several cases where anterior teeth with considerable bone loss and pocket formation were intruded by orthodontic means three or more millimeters into the bone without special periodontic treatment, the bone level receded with the tooth roots. In other words the band of epithelial attachment prevented the clinical root from being increased in depth. But when periodontic treatment was given during the orthodontic intrusive procedure and repeated at twice weekly intervals of a type designed to curet out the epithelial attachment the bone level remained while the root progressed into it thereby markedly increasing the depth of the clinical root. If this is a fact borne out by further study it would seem to have great significance and open up an avenue for further experimentation.

I maintain that it is unnecessary to reduce pocket depths to zero or near it in order to save teeth in health, comfort, and function. Nor is it necessary in most cases, where teeth are savable by any means, to get reattachment of periodontal fibers and reformation of alveolar bone. But these objectives are desirable and must be the aim of continued efforts by periodontists and experimentalists. These ideas (1) that the new line of epithelial attachment is crownward of the old line following a good resection technic, and (2) that the line can be forced crownward by curettage while intruding the tooth, seem to merit discussion.

Discussion

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The initial statement by Dr. Leonard, as in every problem presented in "In Our Opinion," is stimulating, complete and cogent. The material of the present statement presents general knowledge on the subject appropriately and clearly and introduces new experiences of Dr. I. Glickman and of Dr. S. M. Bien. The observations presented by these people are interesting and deserving of more work. Periodontists generally will welcome further information on these interesting topics.

The statement that "the pocket remains as a deepened sulcus" following treatment implies that, when a pocket is free of irritants and inflammation, its depth may be disregarded as a pathologic entity. If one agrees with those, of whom two are Miller[1] and Appleton,[2] who feel that a pocket is a possible source of focal infection disease, continued treatment to insure pocket elimination is necessary. This is done by removing the gingival wall of the pocket by vigorous toothbrushing or other mechanical means, by surgical removal or by reattachment of the soft tissue to the prepared root surface. The last method reduces the pocket to normal or nearly normal sulcus depth with less recession of the gingival margin than would be induced by surgery.

In the instance of focal infection, I feel that the teeth cannot be considered retained *safely* from the standpoint of systemic health unless periodontal pockets are eliminated.

My clinical experience with gingival resection leads to the conclusion that all surgical procedures aimed at pocket elimination result in recession of the gingival margin. In fact, gingivectomy or other surgical procedure is my choice of treatment specifically in those areas where re-

duction of gingival height is desirable. These areas are most frequently the gingival areas classified by Box[3] as those in which deep pockets are likely to develop. They are: narrow pocket on a broad root surface, broad pocket on a narrow root surface, distal pocket on a third molar, mesial pocket on a tilted tooth distal to an extraction space, and a pocket due to failure of the tooth to drift under undue stress.

Another constant observation is that healing following surgical removal of the soft tissue side of a pocket normally permits the crownward growth of epithelium within limits. Epithelium develops above the incised gingiva to form the new gingival margin and its underlying sulcus. This allows for an increase of gingival height beyond the level of the operated area but *always apically to the original gingival level.*

Conservative curettage through the pocket opening following occlusal equilibration and the elimination of inflammation of the tissue side of a pocket often results in pocket healing and subsequent filling in and increased height of alveolar bone.

In discussing wound healing, Boyd states that "the gap is first filled with a mixture of coagulated blood, fibrin, and inflammatory exudate and upon this scaffold the fibroblasts and vascular endothelium build granulation tissue. This process commences at the base and works to the surface."[4]

The problem of maintaining the blood clot is a significant one, as stated in the Initial Statement, yet is present only for the first twelve hours following treatment since, to quote Boyd further, "on an average it may be said that fibroblastic and endothelial proliferation occurs by the end of twelve hours." Furthermore, to quote Boyd again, "epithelium covers the surface by the fourth day and at the end of three weeks there is fully formed non-vascular scar tissue."

Clot maintenance and healing may be facilitated by consideration of a number of related factors. The occlusion should be relieved to obviate mechanical trauma. Toothbrushing should be avoided for the same reason. Irritants and infection should be eliminated by complete curettage. An abundant blood supply should be available by previous treatment including an effective toothbrushing technic. Ascorbic acid should be supplied to stimulate the formation of adequate amount and quality of collagen essential to healing. Finally, the application of a periodontal varnish (such as the formula by Blass in Miller's text) to the blood clot tooth and gingiva of the operated area or the incorporation of thrombin and tyrothrycin with the blood clot aids appreciably in maintaining the clot for the necessary length of time.

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BERNARD S. CHAIKIN, D.M.D.
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Dr. Leonard states that three results occur when pockets are treated, namely: (1) The pocket remains as a deepened sulcus, the free gingival wall being held tightly against the tooth by healthy gingival connective tissue. (2) The gingival wall gradually shrinks down until the gingival sulcus is of normal depth. (3) The lining epithelium of the pocket attaches itself to the root surface thus reducing the pocket to a normal or nearly normal sulcus depth without recession of the gingival margin. He quotes Dr. Irving Glickman who claims on the basis of experimental and histological evidence that the gingival margin after resection heals to a level which is more

crownward and that a more crownward line of epithelial attachment means a greater reattachment of principal fibers with formation of new cementum and a higher level of supporting bone.

In the treatment of periodontal disease we all strive to eliminate the pocket, but wherever it is feasible, to conserve as much of the tissue as possible and still maintain health. The devotees of subgingival curettage, which includes its many variations of removing the pathologic granulation tissue, scaling and polishing of the cementum and dentin, maintain that unless the corium is left, there is no chance for the blood clot remaining so that new granulation tissue may develop and thus at least give the tissues a fighting chance for reattachment. They also feel that reattachment is possible, but only if the walls of the pocket are allowed to remain.

The advocates of surgical resection maintain that a zero pocket is the ideal, but if a regrowth crownward of the cementum, connective tissue, and epithelium takes place, with the sulcus remaining close to zero in depth, it is even more desirable.

Clinically, it is understood that when a pocket is closed so tightly that an instrument cannot be placed between the soft tissue and tooth, that reattachment has taken place. Unfortunately, the only proof which can be relied upon is one shown upon a microscopic slide of the area. As this is not a practical procedure, it will, therefore, have to remain as a clinical observation. It does not follow, though, that because an instrument cannot be inserted between the tooth and gingival margin that reattachment of the principal fibers has taken place. Pockets can be closed by a tightening of the gingivae around a tooth with no biological attachment. This will not prevent a subsequent irritation from causing the gingival tissues becoming inflamed thus producing a wider sulcus which can collect debris and infection resulting in a deeper pocket.

In most cases treated by subgingival curettage, the marginal gingivae will shrink causing the sulci to become much shallower. In fewer cases, when shrinkage does not occur, the depth of the sulcus either remains the same or possibly becomes deeper, but in only a small minority of the cases does the level of the marginal gingivae remain unchanged with reattachment taking place.

In those cases treated by surgical resection, the marginal gingivae will remain low in most cases with a crownward growth taking place to some degree in a small majority of the cases. It has been our experience that when a great amount of crownward growth of the marginal gingivae takes place, reattachment along with this growth rarely occurs.

Glickman and Lazansky cut away bone as well as the gingivae in animals which had no periodontal disease except a mild gingivitis. It would be natural to expect a complete regeneration in these cases. Like results should occur in healthy humans having no periodontal disease when the same operation is performed. We on the other hand, treat cases which have been affected by a slow pathologic process and because of this the amount of regeneration is not usually so great.

In 321 consecutive cases, we found in 182 treated by subgingival curettage, which included many of its variations, that the pockets averaged 5.77 mm. before treatment, that immediately after healing, the sulci averaged 1.11 mm. in depth and that in from six months to five years after treatment, 54% showed an average crownward epithelial growth of 2.5 mm. and 46% showed no regeneration. In 139 cases treated by gingival resection, the pockets averaged 7.64 mm. in depth before treatments, the sulci measured 1.03 mm. immediately after healing, and after a period of from six months to five years, 51% showed an average crownward growth of epithelium of 1.49 mm. with 49% having no regeneration.

The above figures include those cases in

which failures developed where a crownward growth of epithelium occurred with no attachment. These figures also show that if cases are successfully treated, by one method or another, the ultimate amount of healing and regeneration should be about the same.

I agree with Dr. Leonard that teeth around which the sulci depths are greater than zero or near zero can be useful members, but that they will necessarily require more attention with physical care and also more detailed periodic periodontal check-ups.

EDGAR D. COOLIDGE, D.D.S., B.S.
Chicago, Ill.

The function of the epithelial tissue as stated is to "cover and protect the underlying tissue." It is essential that the erupting tooth should force its way through the mouth epithelium but nature has provided the unique method of allowing the tooth to erupt without leaving a break in the continuity of epithelium which we call "the epithelial attachment."

After the tooth has erupted into its occluding position, shall we say that so far the changing line of the epithelial attachment is physiological in character. It is common knowledge from clinical observation that the line of epithelial attachment still progresses rootward on most teeth thereby deepening the gingival sulcus unless the overlying gingiva undergoes some compensating recession. This also may be a physiological change but nevertheless it is a continuous rootward progress of the epithelial attachment as a natural function of that tissue.

When the gingival sulcus deepens it is very evident that it brings about an increased hazard from local irritation due to increased difficulty of maintaining gingival hygiene.

It is logical to my reasoning that the maintenance of shallow gingival sulci is the first principle of treatment and were there no other factors to be considered such as systemic influences the treatment by surgical resection would remove the pocket and the condition would again approximate a physiological state. We know this occurs in a very large per cent of cases treated but does not always stop the continuous bone resorption and downward growth of epithelium because we have not removed the cause of the disease. If the cause of the disease is removed it seems to me there is no reason to expect or anticipate the possibility of reversing the natural tendency of the epithelium to reattach itself where connective tissue has been lost and epithelium has separated from the root surface.

In the treatment of pockets that are too deep to entirely eliminate by surgical means it is often possible to salvage some seemingly hopeless teeth by conserving the granulation tissue in the deepest part of the sulcus which may still contain some potentiality of connective tissue repair. This would indicate that the connective tissue cells have not degenerated and epithelium has not entirely covered that part of the root surface.

In the initial statement the comment is made that the old epithelial attachment is curetted away or cut away in gingival resection.

This statement is misleading and incorrect. I do not think either treatment by curettage or by resection aims to disturb the attached epithelium. In my opinion such treatment would not be indicated. Treatment is intended to reduce the pocket depth but not to destroy the attached tissue. The pocket tissue is only that epithelial lined tissue extending to the line of attached epithelium at the bottom of the pocket.

I do not look for a new line of epithelial attachment after treatment. I try to prevent further progress of bone loss and downward growth of epithelium. The suggestion of a "forced new epithelial attachment crownward" by any manner of treatment will require some substantial histological evidence of proof. Such teeth

cannot be examined by histological methods before and after treatment. How then is such proof to be established?

DR. EDWARD FISCHER
Fairfield, Conn.

It is this discussor's opinion that if the epithelial attachment is not removed, either by so-called "conservative" or by "surgical" means, no "healthy gingiva tightly hugging the tooth" can long remain, if it ever occurs. There is bound to be seepage at the gingival crevice, and eventual ulceration in the inner border of the soft tissue side of the crevice, even though the outer gingiva appears healthy.

Marked ulceration is a common microscopic finding along the crevicular surfaces of gingival tabs which appear healthy by inspection of the labial aspect.

It does not seem that a six month period is time enough to gauge the final results of treatment. The most reliable index we have of successful treatment—in addition to the clinical oral findings, is the x-ray. In order to obtain useful radiographic information, it would seem that a year should be allowed to elapse, before a decision as to success or failure in treatment is made.

In the matter of whether or not it is possible to get reattachment "crownward" as Glickman contends, this discussor has not conducted any controlled clinical or histological experimentation. However, it is my clinical impression that when treatment includes removal of the epithelial attachment, the gingival margin following elimination of the pocket is very often higher than the attachment level of the previous pocket despite the fact that the gingival sulcus is nearly zero depth. In an effort to attain coronal reattachment, it is essential to remove the epithelial attachment, and the lining of the pocket wall, so that it then becomes possible for new cementum and new connective tissue fibers to be reattached. Clinical impressions only are the basis for the above opinion.

It would seem that conditions prevailing in the treatment of parietal abscesses are not pertinent to this discussion of "reattachment." However, some information has been offered concerning the use of foreign substances in the treatment of a pocket, not for the purpose of promoting bone growth, but for the prevention of downgrowth of epithelium. Miller of Louisville suggests inserting acrylic tabs during the post gingivectomy healing in the area distal to the mandibular last molar.

Regarding the concluding portion of the initial statement "Nor is it necessary in most cases . . . to get reattachment of periodontal membrane fibers and reformation of alveolar bone." In the opinion of the discussor, it is not only desirable to obtain these objectives, but it would seem if such results are possible, (and in the writer's opinion, do occur), any treatment whereby they could be obtained would certainly be preferred to half-way measures which yield provisional results and ultimately necessitate elimination of the remaining sulcus.

J. S. FRIEDLANDER, D.D.S.
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The question when may periodontal treatment be considered successful, revolves essentially around reattachment. There are two possible types of reattachment. One is by epithelium, the other by fibrous connective tissue.

That epithelial reattachment is possible has been demonstrated. That it be accepted as a successful treatment is questioned.

There should be no question that reattachment by fibrous connective tissue is more desirable. Whether it can be achieved in a periodontal lesion depends upon the extent of the lesion and the type of therapy.

The potentiality for this type of repair has been confused by a comparative study with experimental animals. There is no basis for comparison of repair in a fresh

surgical wound, uncomplicated by periodontal disease, with that in a chronic periodontal lesion. In the former, the cementum, which is the important tissue of reattachment of the tooth, is contained in a physiological environment and a favorable vascular field. In the latter however, the operation is performed in an infected area The cementum here is contaminated, its cells dead, and the matrix infected.

In an experiment that was performed on animals that had periodontal disease, the classic sequence of repair was not demonstrated.

Whether epithelial reattachment maintains the teeth and their supporting structures in health and reduces the depth of the pocket should be reviewed. It has been shown that epithelium may grow down along a pocket and attach itself to exposed cementum beyond a denuded area at a point where cementoblasts are in contact with intact fibers of the periodontal membrane. The denuded cementum is thus excluded from the attachment. The only reduction of the pocket that may then be expected is that which usually occurs when the edematous tissue becomes firm following the reduction of the inflammation. Some shrinkage usually takes place following adequate tooth brushing.

However if a deep pocket remains, no matter how close the epithelial lining is adapted to the root surface, contamination is a possibility. This poses two important problems: (1) The contaminated tissue fluids may seep through the endothelial cells of the walls of the neighboring lymphatic tubes which drain the area into the submaxillary lymph nodes. These contaminated tissue fluids may act as an irritant to the surrounding connective tissue, including of course the fibers of the periodontal membrane. (2) The effect of the accumulation of toxic material, from many lesions, on the kidneys must be given some thought.

Curetting the old epithelial attachment should improve the possibility of fibrous connective tissue attachment. To enhance the possibility, the lining epithelium as well as diseased cementum should be removed.

Gingivectomy offers the best possibility for reattachment. I believe that unless the basal cell layer of the epithelial attachment is detached surgically, unless "dead" cementum is removed and the soft tissue wall dissected out, this ideal of reattachment is doubtful. The only exception may be in a fulminating infection that is treated quickly. If the cementum is not subjected to prolonged contamination the vitality of its cells may be maintained.

I have treated two such cases conservatively. Both were lower second molars that had deep buccal pockets. Reattachment was complete from a clinical viewpoint.

The principal objection to gingivectomy is concerned with esthetics. Although most cases demonstrate a crownward reattachment, often, in deep pockets there is an absence of the interdental soft tissue. Kirkland occasionally modifies his technique in the anterior part of the mouth. He does a complete gingivectomy on the lingual surfaces of the teeth and a partial resection on the labial surfaces.

Bien's experiment is interesting. There is sufficient experimental evidence of the value of judiciously applied traction as an aid in osteogenesis. It would be of considerable value to follow the changes in the epithelial attachment by taking histological sections during the course of the treatment.

FRANK E. BEUBE, D.D.S.
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There are a number of diverse periodontal technics that have been able to achieve an effect whereby new alveolar bone has formed where there was once deep periodontal pockets because of the loss of the connective supporting tissues.

The presence of this newly formed bone is not indicative that cementum and periodontal membrane have also been generated. Likewise, the position of the epithelial at-

tachment, in a more apical or crownward relation cannot be determined by this evidence. It seems quite probable however, that the epithelial attachment has formed in a more coronal position than the old line of epithelial attachment in order for new bone to have developed. Stating this in another way, that if new alveolar bone formed but without periodontal membrane and cementum, then a long narrow intrabony pocket would have remained, and the epithelial attachment should be formed at its original position towards the apex of the tooth.

There are several technics which are likely to contribute to raising the line of the epithelial attachment. (1) "A well curetted gingival margin"[1] and curettage of the crevicular epithelium and granulomatous tissue lining the periodontal pocket and removal of the existing epithelial attachment might achieve the desired result. (2) Interdental resection may also induce a more coronal position of the epithelial attachment since this method does not excise the entire gingival tissue comprising the periodontal pocket. When regrowth of the interdental tissue occurs, it is commonly found in the same position before this surgical operation was performed. If pocket closure through reattachment was achieved, then the epithelial attachment would be higher. (3) Similar repair can be expected from a flap operation with implantation of cow-bone powder, because the gingival mucosa is kept intact by this operative technic and if healing of bone occurs, the attachment of epithelium may be more crownward. (4) A gingivectomy for the eradication of periodontal pockets which are essentially horizontal, might also contribute to raising the line of the epithelial attachment, especially if no escharotic drug or similar means is used to destroy the newly formed granulating tissue along the wound edge. It has been found by this surgical method that the new gingival margin is more crownward than the line of incision and no measurable pockets are discernible. In other words there is less gingival recession than the amount of gingiva excised. This might mean that the new epithelial attachment is to be found more crownward than the old line of attachment.

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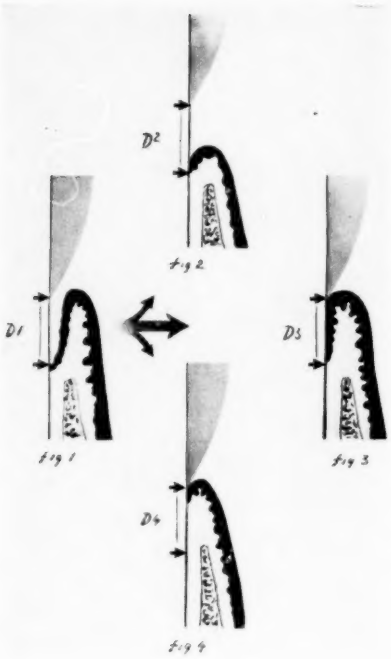
The subject of "reattachment" is an aspect of post-treatment repair of the periodontium which has been documented by numerous controversial clinical and histological reports. There is general agreement regarding the details of the cellular changes entailed in the reparative process following the treatment of a periodontal pocket; however, the question of the exact manner and location on the tooth surface at which the gingival epithelium and periodontal membrane become attached following treatment is the perennial "bete noir" of periodontal circles.

"Reattachment" may be defined as the restoration of gingival contour effected by the re-embedding of periodontal fibres into newly-formed cementum and the attachment of gingival epithelium to tooth surface previously denuded by chronic periodontal disease. The area which is the subject of contention is the portion of the tooth surface denuded by chronic periodontal disease (Fig. 1, Area D^1). In essence, the problem of reattachment hinges upon the question of whether treatment of a pocket will be followed by reattachment of the tissues to this area (Figs. 3 and 4), or whether they become attached at the level of the base of the pre-existent pocket (Fig. 2).

Most of the controversial discussion has been in regard to the periodontal pocket (gingival pocket) whose base is coronal to the level of the alveolar bone (Fig. 1). "Reattachment" following the treatment of pockets in which the base is apical to



the alveolar bone is considered to be a likely possibility even by those who do not accept the possibility of "reattachment" following the treatment of a gingival pocket.

There are two phases to the problem of "reattachment." The first deals with the mechanism whereby periodontal fibres and gingival epithelium become attached to the tooth surface, and the second deals with the location of such attachment. Reattachment of the periodontal membrane entails the embedding of principal fibres in newly-formed cementum. Attachment of gingival epithelium is effected by the basal layer of the epithelial attachment. Box has demonstrated reattachment of the epithelial lining of the pocket (Fig. 3). This type of attachment has been questioned by Orban.

It is generally accepted that if the epithelial attachment and periodontal fibres are artificially detached from the tooth surface, they will be returned to their previous position on the tooth in the course of repair, provided infection does not interfere.

Is it theoretically possible for periodontal fibres and epithelium to become reattached to tooth surface previously denuded by chronic periodontal disease? What are the possible obstacles to such reattachments?

A consideration in the problem of reattachment which is mentioned far too infrequently is the individual systemic background. Systemic disturbances can impair healing. Systemically motivated retardation of the constructive tissue phenomena which are an integral part of repair could conceivably affect the height of reattachment. This phase of the reattachment problem offers a promising approach for research.

Another consideration is the cementum. Changes which occur in the cemental wall of a periodontal pocket include degeneration of the remnants of Sharpey's fibres, accumulation of bacteria and their products and surface fragmentation. Such alterations would prevent reattachment; however, this obstacle to reattachment can be eliminated by careful planing of the tooth surface.

The next theoretical consideration is the clot. In the course of healing, the clot is replaced by granulation tissue. The fibrin of the clot acts as a guide for the proliferating fibroblasts and young capillaries. Organization of the clot by granulation tissue may occur up to the clot surface. Shrinkage of the clot and extension of epithelial spurs along its surface followed by attachment of the epithelium to the root could prevent reattachment of the periodontal fibres. It has been demonstrated that the rapid proliferation of epithelium could be a deterrent to reattachment of periodontal fibres. However, epithelium in the course of healing tends to be guided by the contour of the surface along which it moves. The height of the healing connective tissue, therefore, influences the level at which the epithelium will become attached to the root.

Glickman and Lazansky conducted an

experiment to determine whether removal of the marginal periodontal tissues would jeopardize the height to which the periodontium would be restored in the course of healing. In this experiment[1] the marginal gingiva, epithelial attachment and alveolar bone were surgically removed in dogs and the cementum scarified to a point ¼ inch below the cemento-enamel junction. All of the animals presented some degree of chronic marginal gingivitis at the outset of the experiment. The area of operation was exposed to the oral cavity, without a pack for a 30 day period and then examined clinically and microscopically.

The findings indicated that following gingivectomy scarified tooth surface is receptive to the deposition of new cementum, the embedding of new periodontal fibres and the attachment of epithelium coronal to the level of the line of incision. This experiment did not deal specifically with reattachment to tooth surface denuded by chronic periodontal disease. It did, however, demonstrate that an artificially established gingival level could be added to in a coronal direction in the presence of scarified denuded tooth surface upon which new cementum could be deposited.

Within the limitations governing the applicability of animal experimentation to clinical problems in humans, the findings lend themselves to conjecture regarding "reattachment" following treatment of the periodontal pocket. Thus, if the epithelial attachment and the lateral epithelial wall of the pocket are removed and the tooth surface cleansed of all debris, the periodontal tissues may be reattached to the tooth surface coronal to the level of the base of the pre-existent pocket. The findings also suggest that the formation of granulation tissue and its subsequent maturation can occur in such a manner as to guide the proliferating epithelium to a level of attachment on the root which is coronal to newly-formed cementum and reattached periodontal fibres.

It is of interest to consider the method of dealing with the epithelial attachment in the treatment of the periodontal pocket. If the epithelial attachment is left intact, the maximum post-treatment height which the bone can attain is automatically predetermined because the height to which connective tissue fibres can be attached to the root is established before post-treatment repair occurs. On the other hand, if the epithelial attachment at the base of the pocket as well as the lateral epithelial wall is removed in the course of treatment, conditions are created in which reattachment of the principal fibres, coronal to the previous level of the base of the pocket could occur. It would appear that regardless of whether the surgical or nonsurgical methods of treatment for pocket eradication are used, the epithelial attachment should be removed.

This discussor takes exception to the inclusion of foreign substances in the treatment of the periodontal pocket and would discourage, for the present at least, the consideration of such procedure in a discussion of "reattachment." The only histological evidence thus far available regarding the healing of periodontal tissues in the presence of bone dust deals with areas other than the gingival margin. Such studies are not pertinent to the question of "reattachment." The height to which bone will be restored following periodontal therapy is governed by the height of the connective tissue attachment to the root. This in turn is dependent in part at least upon the maturation of granulation tissue and its differentiation into cells associated with bone and cementum formation. That the presence or absence of bone dust in any way influences the height to which periodontal fibres will be attached to the root has never been demonstrated. Until its usefulness in this connection is substantiated by conclusive histological studies in the region of the marginal periodontium, consideration of use of bone dust in the treatment of the periodontal pocket offers an interesting, but none the less, unsupported speculation.

A certain danger lies in arbitrary posi-

tive or negative declarations regarding the occurrence of "reattachment" following treatment of the periodontal pocket. One is inclined to be somewhat incredulous when confronted by the dogmatic statement that "reattachment" is not possible, if for no other reason than that the present status of knowledge is not definitive enough to warrant such certainty. It is not unlikely that attempts to establish a categorical "yes" or "no" attitude regarding the possibility of reattachment is somewhat less realistic and less consistent with the facts than a flexible intermediate viewpoint which lies somewhere between these extremes.

The problem of "reattachment" is primarily concerned with quantitative rather than qualitative aspects of repair. The possibility of attachment of periodontal membrane and epithelium to the tooth surface from which it has been surgically detached is not challenged. In controversy is the question of whether or not cellular changes identical with those occurring on one side of the line of attachment of a pre-existent pocket can occur on the side coronal to it. It is very doubtful whether the cellular activity entailed in the repair of the periodontium is seriously influenced by a demarcation left on the tooth surface by the base of a no longer present pocket.

It is the opinion of this discussor that if the epithelial attachment and the epithelial lining of the pocket are removed and the root surface is thoroughly debrided of all degenerated and foreign material some degree of "reattachment" will invariably occur in post-treatment repair. Emphasis in the "reattachment" problem should therefore be shifted from controversy regarding whether or not it can occur to considerations of the factors influencing the extent to which it occurs and to exploration of the methods whereby maximum "reattachment" may be obtained.

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The initial statement by Dr. Leonard—raising the line of epithelial attachment and increasing the depth of the clinical root—represents another wording of the reattachment problem. Discussion of this problem in an effort to further research in this field is of utmost importance if technics in periodontal therapeutics are to be based on sound principles. This subject has been approached from many directions, clinically and experimentally, but as yet there is no clear-cut evidence to settle the question, although many new facts are coming to light. The following is a résumé of my concept of reattachment in light of present day knowledge.

First, however, I believe that the term "pocket" should be defined. A pocket may be defined as a pathologic gingival crevice. Another definition is: a pocket is a space lined on one side by the tooth and on the other by an ulcerative crevicular epithelium limited at its base by the epithelial attachment. The main features in the gingiva are inflammation and epithelial proliferation. Calculus is usually seen attached to the tooth occupying the space of the pocket.

Periodontal pockets may be classified as gingival or intrabony, the former being described as one in which the base of the pocket is coronal to the alveolar crest, and the latter as one in which the base of the pocket is apical to the bone crest. The intrabony pocket is not an intra-alveolar pocket, since the alveolar bone of the affected tooth is usually lost. Thus, an intrabony pocket cannot occur on the labial surface of a maxillary incisor, since if bone resorption occurs, a total loss results because of the thinness of the bone. However, although the alveolar bone of the proximal side of an affected tooth is lost, the alveolar bone housing the adjacent tooth may remain intact, resulting in a "V"-like defect. Thus the base of the pocket is apical to the remaining interdental bone crest; this constitutes an intrabony pocket.

Next it is essential to define the term "reattachment." The process of reattachment may be described as a re-establishment of an organic connection between tooth and gingival corium (gingival pocket-production of cementum and organization of connective tissue) or between tooth on one side and bone and gingival corium on the other (intrabony pocket—apposition of cementum, organization of connective tissue and apposition of bone) in an area of a previously existing pocket.

The question as to whether re-establishment of a connective tissue connection to the tooth is possible, in an area of a previously existing pocket, must first be considered. It has been known for a long time that cementum covering of the tooth is easily repaired and new periodontal fibers become incorporated in it. However, it is necessary for a blood clot to form and organize. In order for reattachment to take place it is necessary to remove completely the crevicular epithelium and the epithelial attachment and probably the surface cementum. The above constitutes the basis of the subgingival curettage technic. The hindering factors are the possibility of the downward growth of the epithelium from the outer border to reline the crevice and also shrinkage of tissue after treatment. From a practical point of view while reattachment to a full measure is biologically possible, it is more probable that with shrinkage and the relining of the crevice by epithelium that only the portion at the base of the wound would possibly reattach. In practice one should expect but minimal reattachment by the subgingival curettage technic in the gingival pocket. The amount of reattachment, however, may be dependent upon the accomplishment of the technical procedure. Reattachment as the result of subgingival curettage in the intrabony pocket is governed by similar principles. However, here shrinkage plays no role for the gingival tissue is supported by the bone crest. Also the blood clot is better protected. Many clinical reports in the literature attest to the fact that bone apposition with regeneration of a lamina dura occurs after therapy of an intrabony pocket.

In a gingivectomy technic which eliminates the gingival tissue down to the apical end of the epithelial attachment or beyond, it has always been considered that the new point of attachment of the gingiva is coincident or apical to the original bottom of the pocket; in isolated instances the gingival attachment appears coronal to the original attachment point. This is considered to be dependent upon the blood clot and the patient's reparative ability. One cannot infer, however, that because after gingivectomy occasionally a new line of attachment is more occlusally placed, that one should expect routinely to remove the tissue side of a pocket and have reattachment to a point opposite the previously existing gingival margin. True reattachment may take place in an area directly above the initial incision. Thus, the same factors present in subgingival curettage apply.

It is my opinion that elimination of the periodontal pocket resulting in a gingival crevice which approximates zero and the achievement of a physiologic architecture are paramount objectives in periodontal therapeutics.

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It is a bit difficult to discuss Dr. Glickman's statement without having seen his paper or the evidence he presents, and I am sure that he answers many of the questions that will be raised in the discussion. The question of crownward movement of the epithelial attachment might be solved by notching a tooth at the base of the pocket immediately after complete elimination of a deep pocket and examining the tooth and supporting structures histologically six months later. If an epithelial attachment extends past the notch, the answer is clear.

However, several clinical observations, which are not based on histologic studies, lead me to speculate that the apparent reattachment of newly formed gingivae to roots after gingivectomy is really very close adaptation caused by cicatrization. After removal of a portion of the skin, the granulation tissue becomes highly fibrotic and frequently contracts. Contraction of a ring of new gingival tissue about the root would be likely to cause close adaptation to the tooth.

Although gingivae which have receded after curettage do not seem to grow back as long as they remain in a healthy state, it has been observed by many clinicians that two to six months after complete elimination of deep pockets by surgery, the gingival margin frequently returns part of the way back to its pre-gingivectomy level, slightly reducing the length of the clinical crown. In some cases, this regrowth of gingival tissue may result in a partial or complete reformation of the pocket, but in most cases it is difficult or impossible to pass an instrument into a crevice. It is possible to prevent the recurrence of pockets by insisting upon adequate brushing and gingival stimulation, by correcting occlusal trauma and other etiologic factors, by providing knife-like tapering contours in the gingivectomy, by eliminating broad shelves of tissue, by avoiding abrupt rises and falls in the outline of the incision, and by doing osseous resection when necessary. However, when the line of incision dips into the areolar mucosa (in cases where extremely deep pockets must be eliminated or where the areolar mucosa extends relatively close to the gingival margin) there is a considerable likelihood that regrowth of the gingivae will result in formation of a new crevice. On the other hand, in cases where the incision remains in tissue that is firm and pink, regrowth of the gingivae is less likely and any new tissue that forms seems clinically to be attached to the tooth.

It has been my impression that in certain cases where even the marginal and alveolar gingivae seem to be less highly keratinized and less fibrous than normal and slightly darker in color, there is a greater tendency for regrowth of tissue and return of part of the original crevice than in other cases where the marginal and alveolar gingivae are pale pink, firm and more definitely stippled. While there is undoubtedly some kind of systemically-produced defect in the former type of tissue, it would seem as though it and areolar tissue are both less fibrotic than normal marginal and alveolar gingival tissue and cicatrization might therefore be less extensive. To follow this line of reasoning further, insufficient cicatrization would cause newly formed gingivae to lie flabbily against the root surface and permit instruments to be introduced between it and the root.

Dr. Bien's interesting cases bring to mind a rather similar one in which a 45-year old woman with fanned-out separated anterior teeth with about one-half of the normal amount of alveolar bone remaining, was treated orthodontically with great success. The anterior teeth were moved lingually and intruded slightly and are now close together and in good alignment. The bone now is considerably nearer the normal height and it is not possible to enter the formerly deep proximal and palatal pockets. It was not ascertained whether the increased alveolar height was due to the intrusion of the teeth or to formation of new bone, but in this case the periodontal treatment was completed before the orthodontic procedure was started and no periodic curettage of any kind was performed while the teeth were being moved. This unusual case is described in order to suggest that the increased alveolar height in Dr. Bien's cases and the periodic removal of the epithelial attachment might have had a coincidental, rather than a cause-and-effect, relationship. A greater number of cases would undoubtedly clarify the point and very valuable information might be derived.

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Dr. Leonard says "that when a pocket is treated and relieved of all irritants and inflammation without appreciably disturbing the band of epithelial attachment, one of three results may occur."

(1) He says "the pocket remains as a deepened sulcus, the free gingival wall being held tightly against the tooth by healthy gingival connective tissue."

I beg to differ with Dr. Leonard in his opinion concerning post-operative results in the treatment of periodontal pockets, presuming that he used the subgingival curettage technique. In my opinion, had he selected gingivectomy as the method of procedure, he would have had the assurance of uniform postoperative results. I think it is much better practice to remove surgically the diseased epithelial band of attachment along with other irritating factors, and then, rely upon the physiological regeneration of a new and healthy epithelial band of attachment. With clean surgical removal of gingival tissue and careful post-operative attention following the gingivectomy technique, there is, in almost every instance, assurance of physiological replacement with firm healthy gingivae.

In my opinion, so long as a pocket remains, although it may be apparently free from inflammation, the lesion, nevertheless, is there and wherever a pocket exists, it surely harbors a certain amount of bacterial matter. It make no difference how tight the band of epithelial attachment may hug the root surface, it must be remembered that bacteria are microscopic in size and capable of penetrating a tight gingival margin. The larger and deeper the gingival sulcus the more bacterial matter it is capable of retaining. Where complete healing and regeneration of a new cortical layer of bone has not occurred in the base of the gingival sulcus following treatment, regardless of the method used and despite the fact that acute inflammatory symptoms have been reduced, it is almost certain that disintegration, though retarded in its progress, continues in a milder state of activity, and, in my opinion, the periodontal lesion has not been cured.

(2) Dr. Leonard says "the gingival wall shrinks down until the gingival sulcus is of normal depth."

Since there is no reattachment of the gingivae to the root wall, and shrinkage is more or less inevitable, why would it not be simpler to remove the over-lying tissue by incision, and quickly eradicate the offending factors involved in the lesion?

In answer to Dr. Leonard's third question, "The lining epithelium of the pocket attaches itself to the root surface, thus reducing the pocket to a normal or nearly normal sulcus depth without recession of the gingival margin."

I fully agree with Dr. Orban in his viewpoint regarding reattachment of the epithelial lining to the root surface. In my opinion, it is impossible to bring about reattachment of the epithelial lining of a pocket to the denuded root surface of a tooth.

There can be no doubt about crownward development of the epithelial band of attachment in the surgical treatment of periodontal lesions by the gingivectomy method. I fully agree with Dr. Glickman in his views in this particular. I have seen evidence in his experimental research work, showing evidence of crownward development following the gingivectomy operation.

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The "Initial Statement" by Dr. Leonard is very interesting. It brings up numerous problems and stimulates further research. I still contend (as stated by Dr. Leonard) that reattachment of the pocket epithelium to the surface of the tooth is a biologic impossibility. Clinical observations alone

cannot be used as proof of reattachment, because we cannot go back and find out the actual conditions. Clinical claims remain as such and should not be used in scientific arguments.

The scientific evidence brought up by Dr. Leonard in favor of a reattachment of the pocket epithelium to the root surface (Harold Box's case and Reppeto's case) are very good, but have to be explained differently. Box never claimed or has shown that pocket epithelium can reattach itself to the root. He claimed that epithelium can reattach itself, but this epithelium is not the pocket epithelium, but the proliferating epithelium of the wound surface, following removal of the epithelial lining of the pocket. He writes: "Following the removal of the epithelial lining and the underlying granulation tissue with the periodontal curettes, the soft tissues are given a final, gentle freshening of the surface which induces further bleeding." Further, "The fibrin fibrils of the clot are closely adherent to the curetted connective tissue," and "A well curetted gingival margin tends to delay the epithelial ingrowth." Whenever Box speaks of epithelial reattachment, he always means attachment of the newly-developing, proliferating epithelium, and not the old pocket epithelium. I find not one single statement in Box's article which could be used against my own view. I agree wholeheartedly with his dissertation on the subject.

As far as Repetto's case is concerned, I have seen his pictures years ago and I am indebted to Dr. Leonard for sending them to me for inspection, so as to refresh my memory. Reppeto's case does not prove in any respect, a possibility of reattachment of the pocket epithelium to the root surface. In this case, the epithelium is attached to dentin. The cementum was removed by curettage. This dentin exposure might have occurred above or below the original level of the bottom of the pocket at the time of operation. There is no proof offered as to which occurred. In either case, not the old pocket epithelium, but the proliferating epithelium of the gingiva attached itself to the dentin. That such an attachment is possible, I never denied, in fact, claimed that attachment of epithelium can only occur by gradual creeping growth of the basal epithelial cells along the root surface (as stated by Leonard). Similar epithelial attachments to dentin were shown by Gottlieb and Orban,[1] Kotany;[2] to bone, by Schoenbauer;[3] to an odontoma erupting into the oral cavity by Wolf.[4] Such findings are extremely interesting from the biologic point of view, but are no proof for a possible attachment of the pocket epithelium to the root surface. The reasons why this is considered impossible were discussed in a paper not very long ago.[5]

As far as a "crownward growth of the epithelial attachment as well as marginal gingiva" after gingivectomy of a certain type, is concerned, the evidence (and it should be good!) has to be presented.

The same holds true for the orthodontic cases presented for discussion by Leonard. Without pre-operative and post-operative casts, pictures, roentgenograms, no one can discuss this problem intelligently, and perhaps not even then!

I still maintain that the shallower a pocket (sulcus) is, the better (zero is ideal, as claimed by Gottlieb), but I agree with Leonard that it is not always necessary to reduce the depth of a pocket to maintain periodontal health. It is only desirable. Our objectives in treating a given case have to be guided by the individual conditions rather than by unchangeable rules.

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I am of the opinion, in accord with the initial statement, that the crux of periodontal therapy depends upon the proper consideration of the epithelial attachment. It grows apically not so much at the expense of the principal fibers but because they have become degenerated and the intercellular substance more fluid. This degeneration seems to occur secondarily as the result of alveolar bone resorption with inflammation following the course of the blood vessels as in periodontitis or primarily as in periodontosis.

I am not in agreement that it is unnecessary to reduce pocket depths to that of a normal sulcus. While it is true that the free gingival wall of a deep pocket is held more closely to the surface of the root after the removal of irritations the condition of the connective tissue of that portion of the gingiva is not comparable to that of the gingiva in a normal sulcus. The principal fibers of the free gingival and transeptal groups are missing and consequently the free gingiva with a deep pocket cannot be as resistant to subsequent injury and accumulation of irritants as that of a normal sulcus.

The continued downward growth of the epithelial attachment in periodontal disease must, in my opinion, be controlled by reduction in the depth of the pocket by means of curettage and subsequent stimulated shrinkage of the gingival wall or by removal of the gingiva including the epithelium, to the bottom of the pocket, in toto. Any attachment or reattachment of epithelium to the surface of the root must take place by the cells of the deeper layers of epithelium which is probably accomplished by mechanical fixation of the tono fibrils onto the rough surface of the cementum. The possibility of reattachment of epithelium after subgingival curettage depends upon the removal of the surface epithelium of the gingival wall. In case of gingivectomy this epithelium is removed and the attachment either remains at the bottom of the pocket or if the epithelial attachment is also removed, becomes reattached at this same place. Whether or not the healing epithelium attaches itself more coronally to the bottom of the pocket, depends upon several factors: (1) The positive retention of the blood clot, (2) The condition of the cementum and (3) The general health of the patient.

It is true that the primary purpose of squamous epithelium is to cover connective tissue and whether it grows *over* or *under* the blood clot must be a matter of clot fixation and delicate timing. The clot must be retained intact sufficiently long for it to become organized, infiltrated with endothelial buds and young fibroblasts. The cemental wall must also be prepared to allow possible histo-differentiation of young connective tissue cells into cementoblasts. If this occurs in time to allow the proliferation of epithelium over the organizing blood clot, attachment of the epithelium more coronally and new connective tissue attachment to the root surface is possible. One can even conjecture that in this event the proliferating epithelium might act as an organizer stimulating the young connective tissue cells to differentiate much as Hertwig's sheath probably does in the development of the root.

I have observed this suggested higher level of gingival attachment after gingivectomy clinically and in my experience it has occurred most often when treating deep lingual pockets of the upper anterior teeth. Here with the increase in anterior posterior bone width due to the confluence of the alveolar and palatal bones, the ease of visual access and mechanical clot protection, the above factors are more readily marshalled. The same conditions do not obtain in interseptal pockets and I would

not expect the same frequency of higher attachment in these areas unless our technic can be materially improved.

The observations made by Saul M. Bien of New York would seem to follow this same pattern. It is obvious to me that so long as the epithelial attachment remains intact the length of the attached root would remain the same during intrusion but if proper periodontal treatment including removal of the epithelial attachment and preparation of the cementum were instituted during intrusion one would simulate the biological conditions obtaining during the eruption process and consequently expect an increase in the length of attached root and in the height of the alveolar bone.

Dr. Leonard's initial statement points out the direction which our future research and experimentation in periodontal therapy should take and we must be forever alert to all possibilities.

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The report of I. Glickman, that the new line of epithelial attachment is crownward of the old line, presents an encouraging advancement in the field of periodontology, and we hope his findings can be affirmed by further scientific investigation. While I have had many successful cases where there has been a definite crownward advancement of the epithelial attachment, it is not too evident clinically and it does not seem that this always occurs, or can be predicted in advance. The interproximal and vertical types of periodontal pockets present a good possibility of reattachment or closure, and the noticeable results are in this category.

The upper first and second molars with trifurcations definitely involved present a periodontal problem. A crownward advancement of the epithelial attachment has been obtained in the following manner. The teeth are given a meticulous curettage on both the hard and soft tissue sides of the pockets. If the trifurcation remains open, the procedure is repeated until the areas have had three complete treatments. Having failed one may then choose a gingivectomy. With a few rare exceptions my only success has been with old chronic ulcerative gingivitis cases. These seem to respond with a fair degree of regularity in mild trifurcations. The most encouraging type of treatment has been the extraction of the tooth with the greatest trifurcation involvement. This gives access to the remaining one permitting a very thorough curettage. The whole area including at least one adjoining tooth on either side is covered with one piece of dry foil completely over the occlusal surface. The dry foil is left in position for one week. The trifurcation closes in many of these cases preventing the entrance of a probe. In fact one of these cases had a closure on both the right and left second molars after the extraction of the first molars. A fourteen tooth one piece fixed bridge was constructed replacing the right and left first molars, and the five anterior teeth previously lost. X-rays reveal a normal trifurcation in the second molars which do not permit the entrance of a probe. It has given two and one half years of satisfactory service to date without any signs of regression.

F. E. Beube in his article on "Interdental tissue resection," *American Journal of Orthodontics* and *Oral Surgery*, June 1947, reports a chart showing a definite decrease of the depth of the periodontal pocket with the interdental resection technic. I do not have any scientific data on the crownward position of the epithelial attachment after gingivectomy. There seems to be no outstanding clinical indication except in the cases cited. Since I. Glickman has reported in the affirmative, it would make a good research project.

H. J. Leonard stated, "It is unnecessary to reduce pocket depths to zero or near it in order to save teeth in health, comfort, and function." This is true in most cases, and many of us follow the conservative treatment in all or part of our cases. There are, however, about 20% of the patients who through financial reverses

or for a number of other reasons fail to report for the three to six month recall visits. The deepened sulcus then becomes a periodontal pocket and our treatment has been in vain. There is also another possibility that one should consider in the patient who has a recurrence of his periodontal condition. That is a transient bacteremia in the rheumatic heart patient causing a sub-acute bacterial endocarditis.

Since greater possibility exists of a future pathological condition developing in a deepened gingival sulcus than with a shallow crevice, the ideal is a shallow sulcus. To attain this end we should use all the available technics: "Interdental Resection" and also "Bone Powder" as advocated by F. E. Beube, the modified "Black Technic of Gingivectomy" as taught in Columbia University, "Osseous Resection" as given to us by Saul Schluger and "Gingivoplasty" sponsored by H. Goldman.

Concluding Statement

HAROLD J. LEONARD

It seems to me that much of value has come from this discussion and I am well pleased with it. Not only have new ideas been presented, but lines of research have been suggested which may make clinicians come closer together in the approach to their goals. The principles of periodontia procedure should be as standardized as those of general surgery or of operative dentistry. They are not now, as shown by Coolidge's statement regarding the undesirability of curetting the epithelial attachment in resection operations, compared with the statements of Beube, Fischer, Friedlander, Glickman, Kirkland and Ray who evidently curet it as a routine. The divergence of opinion regarding reattachment of epithelial lining and of the menace of a deepened sulcus are other matters which should be corrected by research. Since clinical research on private and hospital patients and on animals is impractical, I suggest a follow up of the method used by the California Stomatological Research Group, that of using convicted prisoners as experimental subjects.[1]

With Friedlander's statement that success in treatment revolves essentially around reattachment, I do not agree. The criterion of success in treatment to me is the restoration to health, function, comfort and beauty of the teeth and mouth and the maintenance thereof. Reattachment and the rebuilding of periodontal attachment and alveolar bone are desirable objectives but not essential to success in treatment. Periodontia is difficult enough in its technics to the aspiring dental student without setting up unnecessarily high goals.

Some comment should be made about dead cementum and the loss of vitality of its cells. The cementum is an inert, calcified substance deposited about periodontal fiber ends for the purpose of attaching them to the root surface. Fine canaliculi run through the cementum, evidently for the purpose of keeping it moist and elastic. Where cementum is formed rapidly cementoblast cells become incorporated in it and as they get buried more and more deeply they lose their vitality and disintegrate. Cellular cementum is present toward the apex and in bifurcation areas. The cementum of the gingival half of the root is usually free of incorporated cells. Such a layer of inert intercellular material should not, in my opinion, be called vital or non vital whether or not living tissues are attached to it. The criterion of whether living tissues may again attach to long denuded cementum is determined, as I see it, not by the length of time the pericementum

has been detached from it, but rather by its porosity and the toxic bacteria, fungi and other debris which fill the pores. When denuded cementum is gently shaved to remove the outer porous layer which is filled with living micro-organisms, a surface is left which is relatively non-porous and free of irritating material. There is reason to think from experiments on cementum formation in partially filled root canals by Clyde Davis[2] that such an old denuded but properly shaved cementum would not interfere with new connective tissue formation about it and deposition of new cementum upon it.

Systemic chemical conditions may presumably play a part so that a result obtained in one case might not be applicable to all cases. However in my opinion too much emphasis should not be placed on systemic chemical factors, important as they undoubtedly are in a few cases. Cases where good healing does not occur when adequate surgical conditions are met are fortunately fairly rare.

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1. Stanley, L. L.: J.A.D.A. **12**:161-5 (1925).
2. Davis, W. Clyde.: D. Summary **41**:482-92, 563-68, 787-90 (1921).

Editorial

Preventive Care vs. Heroic Therapy

THE EMPHASIS in periodontal therapy should be changed. The unfortunately too prevalent notion that periodontal treatment is a composite of complicated, essentially heroic technics designed to result in "tightening" almost hopelessly involved teeth should be replaced by a new, mature concept of maintenance of oral health by preventive treatment. The latter is an approach to the maintenance of the health of all of the periodontal tissues in relation to all the teeth of all patients. The concept of "preventive care" offers a constructive viewpoint for the periodontist instead of one of despair. It presents existing periodontal treatment technics as tools for the maintenance of the health of all the periodontal tissues rather than as spectacular steps for "saving" individual teeth. It is emphasis upon the latter aspect of periodontal service which has annexed the stigma of futility to periodontology in the minds of dentists and public alike.

"Preventive care" should be part of the dental service to be rendered all patients instead of the needs of only those with marked periodontal disease. The concept of "preventive care" eliminates from the mind of the student a differentiation between "periodontal patients" and other types of patients. For

patients without notable periodontal disease, "preventive care" is a prophylactic measure; for patients with early periodontal disease, it is effective treatment for the elimination of disease and prophylaxis against tooth loss; for those with marked periodontal disease, it focuses attention upon teeth to be retained and their future usefulness rather than upon those to be lost. Above all, the concept of "preventive care" inculcates a periodontal consciousness into the thinking of the dentist and dental student—a need for eliminating all potential causes of periodontal disease in all patients, and caution against creating conditions which favor periodontal disease. It specifically directs the dentist's attention to a clearly defined phase of general dentistry. It impresses upon him that he is being equipped with the "know-how" for the maintenance of oral health, for the prevention of tooth loss—which if applied early and consistently offers a fruitful outlet for his efforts in both terms of services rendered the patient and personal gratification.

It is with the concept of "preventive care" as a major phase of general dentistry as a background that the question of the time to be allotted periodontology in the undergraduate curriculum should be considered. There are four aspects to the teaching of periodontology:

1. The application of basic sciences to the clinical aspects of the periodontal problem.
2. The presentation of treatment methods and techniques.
3. A crystallization of the interrelationship of clinical periodontology with the other branches of dentistry.
4. Inculcating the periodontal viewpoint into the thinking and over-all clinical practice of the student.

All this requires time. If 66.92% of the dental curriculum is devoted to clinical subjects, wherein does the logic lie in devoting only 2.10% of this to periodontology? More specifically, on what basis is the 48.62% of the dental curriculum allotted to prosthodontia, operative dentistry and periodontology, divided so that periodontology receives only 2.10%. The total time allotted prosthodontia, operative dentistry and periodontology should be re-allocated so that each branch receives an equal share.

It strikes a somewhat hollow note if a student is taught the principles of periodontal treatment and then allowed only enough time to apply them for selected patients, on a restricted schedule, while he piles up a most enviable record of fillings and bridgework in patients whose periodontal needs go unattended because curriculum time, or monumental departmental requirements in operative dentistry and prosthodontia do not permit otherwise. Inculcating the periodontal viewpoint in the student thinking means convincing him that periodontal care is a health service which is a basic part of the general practice of dentistry, to be administered at all times it is needed and that no patient is completely cared for if his periodontal needs go unattended.

Abstracted from a paper by Dr. Irving Glickman, of Tufts College Dental School, and read before the Periodontia Section of the American Association of Dental Schools, March 28, 1950.

Announcements

The Division of Graduate and Postgraduate Studies at Tufts College Dental School announces the following refresher courses in Periodontology:

Periodontology
Each Monday for the Twelve Weeks from
Feb. 12 to May 7, 1951
DR. IRVING GLICKMAN AND STAFF
Tuition $175.00—Class limited to twelve

Periodontology—Occlusal Equilibration
Each Monday for the Five Weeks from
Jan. 8 to Feb. 5, 1951
DR. IRVING GLICKMAN, VISITING CLINICIANS AND STAFF
Tuition $100.00—Class limited to fifteen

Surgical Pathology of the Oral Cavity and Adjacent Structures
Each Thursday afternoon for the Eight Weeks from
Oct. 5 to Dec. 7, 1950
DR. IRVING GLICKMAN AND STAFF
Tuition $100.00—Class limited to ten

Periodontology
One week—June 4 to June 8, 1951
DR. IRVING GLICKMAN
Tuition $100.00—Class limited to twelve

Periodontology
One Week—May 14 to May 18, 1951
DR. OLIN KIRKLAND
Tuition $100.00—Class limited to twelve

The Postgraduate Studies Extension Division of the University of Illinois College of Dentistry, 808 S. Wood Street, Chicago 12, Illinois announces the following course:

OCCLUSION IN PERIODONTAL PRACTICE

THURSDAY AND FRIDAY, FEBRUARY 8 AND 9, 1951,
immediately following Chicago Mid-Winter Meeting

Thursday, February 8, 1951

Essayist's include Drs. Balint Orban, J. P. Weinmann, Harry Sicher, B. H. Downs, Clyde H. Schuyler and John R. Thompson

Fee $25.00

Complete Reprints of "Occurrence of Gingivitis in Children" Available

To conserve space, several tables and charts were omitted from the article "Occurrence of Gingivitis in Suburban Chicago School Children by Drs. Massler, Schour and Chopra which was published in the July 1950 Journal of Periodontology (page 146-164). The reprints of this important artice are complete, however, and may be obtained free of charge by writing Dr. Maury Massler, 808 South Wood Street, Chicago 12, Illinois.

The Colorado Dental Foundation announces the formation of a Histopathology Study Club which should prove valuable to periodontists. Any member of the club is entitled to submit biopsies and then receive slides and a report of the microscopic findings. For further information about this interesting project, write directly to the Colorado Dental Foundation, 629 North Nevada Avenue, Colorado Springs, Colorado.

For several reasons, including a four weeks' illness of the editor, this issue of the Journal is late and smaller than usual. The January 1951 issue, however, promises to be an improvement in both regards. Incidentally, the editor would like to publish short "case history" reports, which emphasize practical *technics*. Why don't *you* send one in?

Books Received

Textbook of Periodontia (Oral Medicine), by Samuel Charles Miller, and members of the Periodontia Staff of New York University College of Dentistry. Third edition. The Blakiston Company, Philadelphia. 900 pages and 587 illustrations.

Endodontia (The Clinical Pathology and Treatment of the Dental Pulp and Pulpless Teeth), by Edgar D. Coolidge. Lea and Febiger, Philadelphia. 300 pages and 180 illustrations.

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